ACUTE INJURIES OF THE HEAD

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ACUTE INJURIES OF THE HEAD

THEIR DIAGNOSIS, TREATMENT COMPLICATIONS AND SEQUELS

BY

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THIRD EDITION

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TO

PROFESSOR GEOFFREY JEFFERSON
TO WHOM BRITISH NEURO-
SURGERY AND MANY YOUNG
NEUROSURGEONS OWE SO MUCH

Made and Printed in Great Britain

PREFACE TO THIRD EDITION

THE 1939-45 war has now so long passed that it is possible to view the surgical activities of that period in dispassionate perspective in particular let us review the part that neurological surgery played in the succour of the wounded not only on foreign fields but on the home battle fronts

I remember vividly the Meeting of the British Neurological Surgeons at Oxford in 1939 peace was in obvious danger and our thoughts turned excitedly to preparations of war, British and American neurologists were discussing the advisability of their attending the coming International Neurological Congress in Copenhagen We know how many did, in fact cross the North Sea to return precipitately at the suggestion of His Majesty's Government before even having read their papers. What a haul of clinical scientists the Nazis could have had, had they so desired !

I remember the commotion at the International Neurological Congress held in London when the venue for the next meeting was being discussed and I like to believe that it was I who suggested indirectly to Gordon Holmes that the Ayes to go to Copenhagen should sit on one side of the hall thereby rendering the counting of the votes more easy than a show of hands We little realised then what heavy days lay ahead of us I wonder what would have happened had by chance Paris been chosen as the meeting place and not Copenhagen Would the Nazis have been as considerate as they seem to have been in Denmark ?

Britain at the outbreak of war was fortunate in having the services of Jefferson Cairns and Dott Sir Hugh Cairns (Nuffield Professor of Surgery at Oxford) was given charge of the Neurosurgical Services of the Army Professor Jefferson was made head of the Neurosurgical Services of the Emergency Medical Services in England and Professor Dott head of a similar organisation in Scotland

When the time came and the right circumstances developed these three men threw out broad opportunities that younger generations readily accepted and with initiative and skilled hands put to excellent use as the results tabulated in a later chapter will testify

The most important decisions in planning resulted from the fundamental belief that to obtain the best results in the treatment of open head wounds, early and complete surgery is essential. It was this conviction that led to the formation of the mobile neurological teams (M.N.S.U.) and the main credit for this is due to Cairns Willie Henderson was put at the head of the first team and it was he who worked out details of tactics Together with George Smyth (neurologist) Martin Nicholls (assistant neurosurgeon) Captain Travener and John Challis (anæsthetist) he took out a team to Flanders in 1940 Later in that year this team stood by 800 of their wounded patients and were taken prisoner and spent years of captivity in Germany There

was ample time for them to escape had they so wished, it was the sense of their responsibility that kept them at their posts When the war was over and these men returned—Henderson to Leeds, Smyth to Manchester and Nicholls to Glasgow and later to Aberdeen—it was soon seen that their experience had failed to leave on them a single psychoneurotic mark Accounts of the work of later teams can be perused in the very interesting and informative chapter, written by Hugh Cairns, in the War Surgery Supplement No 1 of the *British Journal of Surgery*

At the beginning of the war England and Wales were divided into eleven medical regions, Scotland forming a separate zone Each region was given a neurosurgical service These neurosurgical services were often combined with neurology, and in all instances psychiatric co-operation was close The duties of the neurological surgeon were to treat, house and rehabilitate the head-injured of air-raid casualties, to help the medical organisations of the three Services when necessary, and to give general neurosurgical services to their region in the time that was left over to them In order to handle the casualties of the North-West European campaign, three experienced neurological surgeons (Northfield, McKissock and O'Connell) were drafted to strategic points in the South of England

By the nature of things the neurosurgical service of the E M S has had little publicity, it has, however, been happy to regard itself as the silent service

The Canadian Neurosurgical Unit was based at Basingstoke At first it was under the surgical guidance of William Cone of Montreal, a firm believer in early complete surgery However, most of its active work was done when Harry Botterell of Toronto was at its head Of the Canadians, much was naturally expected, and we are indebted to them for other things besides their surgical skill and ability to organise, they came early and without stint when this country was in trouble, to us here it was a tower of strength to see them already lined up during the inactive and trying period of the "twilight" war

Let us now consider the advances that were made in neurosurgery and the lessons to be learnt The fundamental importance of early complete surgery has again been made obvious, indeed, the excellence of the neurosurgical results were largely attributable to this policy

Jefferson and Cairns were both convinced of the value of the academic approach to practical problems, indeed, the ready co-operation of pure and technological scientists was one of the outstanding features and successes of the past war

Blood transfusion not only saved lives but materially lowered morbidity rates by raising a patient's resistance before infection could get a foothold From figures to be given later, it will be seen that a large percentage of open head wounds were firmly closed at the first débridements and that primary healing was obtained in most instances To what extent this success was due to chemotherapy cannot be known with certainty, but to judge from first principles and past experience

it must have been considerable On the other hand it must be appreciated that chemotherapy loses much of its efficacy if the surgical excision of a wound has been inadequate or faulty When infection has once become established chemotherapy is life-saving

Correct rehabilitation and formal psychiatric assessment has led to a higher percentage of recoveries and to speedier return to duty or to work than hitherto I believe that rehabilitation is an essential part of the treatment of the head injured and that without it the long term results of closed head injuries will remain unsatisfactory

The problems of peace time however are very different from those of war In war the surgeon is largely concerned with the prevention and treatment of infection and with the repair of holes in the head As a result surgical publications relating to the war period have been largely concerned with the nature treatment complications and sequels of open wounds of the head. Relatively little has been written on the closed head injury and it is with this subject that this book is primarily concerned. I should like therefore to set out the problem as I see it and to attempt to dispel some current misconceptions

The problem has the following aspects —

1 The physics of the injury
2 The period during which the patient s life is in danger
3 The stage of convalescence
4 Medical rehabilitation
5 Industrial rehabilitation.
6 The final medical assessment and the advice to the patient regarding his return to work.

Although on the physics of closed injuries good work has been done no new concept has emerged That the neurones could be damaged by movements of the brain in relation to the skull was known long before the last war The problem has become largely one of pure mathematics, and further experimental studies with new methods *of inflicting trauma are unlikely in themselves to throw new light on* the problem of concussion

The neurological complexes and the physical injuries of the brain that result from the forces of rotational and linear acceleration are indistinguishable from those that arise from slow squashing deformations of the head such as occur in the mines from falls of coal. The essential force that causes concussion is distortion. Also it must be clearly understood that the lesions of an acute head injury are not those *merely of commotio cerebri or of concussion*—or *better of diffuse* neuronal injuries Diffuse neuronal injury is usually one of the phenomena resulting from injuries of the head and very often in itself does not cause death

By definition concussion means unconsciousness that has resulted from cerebral trauma It should mean nothing more it does not connote any known or hypothetical neuronal pathological state that can account for impairment of consciousness or for the many other *possible abnormal neurological signs* The term commotio cerebri

on the other hand, implies the existence of a lesion, although its precise nature is not known.

At autopsy on patients who died from head injuries it is often obvious that gross lesions such as contusions and hæmorrhages cannot account for the neurological signs that were observed clinically. Therefore, disordered function must have occurred in neurones distant from areas of damage of the degree that can be seen by naked eye. We know little of the nature of these neuronal changes; we likewise know nothing of the phenomenon where a reversible injury becomes irreversible and we are uncertain as to the exact anatomical site of neurones whose disordered function gives rise to the variegated neurological signs. It is to the solution of these problems that I would call the attention of those who wish to do research work on head injuries. Enough has been said on the physics of injury. To medical observers other than neurologists I would point out how fascinating it is to sit by the bedside of a man with a head injury and witness the return of consciousness. The time has now come when we should try to become a little clearer in our minds on what we think of unconsciousness; is unconsciousness a positive neurological condition or is it merely a negative one implying an absence or impairment of consciousness?

To return to the therapeutic point of view, the acute head injury problem is by no means that merely of diagnosis and treatment of diffusely scattered microscopical neuronal injuries. The acute head injury results in a variable combination of pathological states, some of which in themselves are lethal but which are often amenable to surgical or to active medical treatment.

That the subject of the acute head injury concerns others beside the neurosurgeon is one of my main themes; everyone, from those interested in the link between the brain and the mind to those who have to use their skill, administrative or technical, in the prevention of accident, must be recruited in the solution of the many problems that arise.

On more than one occasion, and in more ways than one, scholars of the past have reminded us, as did Santayana, that "Those who cannot remember the past are condemned to repeat it." A study of past experiences can help in two ways in planning for the future, negatively and positively. By negation we know what errors not to repeat. There is, however, a danger in viewing the past to come to negative conclusions only, when more harm than good can accrue through the omission of imagination and of anticipation. We must try to foresee what future requirements will be; we need to know more than what to avoid; to know what to do should be our aim. In any future war may not the artificial sunlight lamp, for example, be as important as the scalpel?

Now to the pleasant task of thanking all those who have helped in the production of this third edition.

My neurosurgical unit was born and grew under the goodwill of my friends Dr J. A. Charles, of the Ministry of Health, and Professor

F J Nattrass Professor of Medicine at the Newcastle-upon Tyne Medical School. To these two gentlemen neurological surgery of the North East owes much The surgeon cannot do his best without encouragement and opportunity I have received both in good measure from both these friends and I thank them

I should also like to take this opportunity of saying good bye to the Medical Officers of Health of the North East Region through whose ready co-operation I was able to concentrate my work in one clinic, in particular I would mention Dr Walton Medical Officer of Health of Newcastle-upon Tyne Dr McCracken, Medical Officer of Health of the County of Durham Dr Tilley Medical Officer of Health of the County of Northumberland and Dr Grant Medical Officer of Health of Gateshead. I am also indebted to Dr Hurrell, Medical Superintendent of the Newcastle General Hospital for his helpful co-operation

For the new coloured photographs of the pathological specimens my thanks are due to Mr Dudfield Rose of the Newcastle Royal Victoria Infirmary I have also to thank Mr Gordon Hilton for his clinical photography

I am again indebted and grateful to my friend Dr Raymond Whitehead of the Department of Pathology of the University of Manchester for his help and criticisms in the production of this edition

Miss Bousfield has helped me with each edition of this work and much credit is due to her It would be difficult to overstate her part in the productions

Once again my warm thanks are due to Mr Macmillan for his understanding and encouragement

In the past I have been granted good facilities for recording observations and some for research I still need more of both

Finally I would gratefully acknowledge the help received through reviews of the second edition of this work I hope that I have profited from them and shall always welcome constructive criticism

G F ROWBOTHAM

FAIRHOLME,
WYLAM.
1949

PREFACE TO SECOND EDITION

I BELIEVE that the next advance in the treatment of civilian head injuries after the war will be the closer co-operation between the general practitioner and the specialist, between the specialist and the Government and, finally, between the Government and industry.

This war has proved beyond doubt what some of us already knew, that for good results and early readjustment following a head injury, a man or woman must have continuous and co-ordinated treatment from the acute stages of injury until he or she has been finally resettled in employment, either in industry or in the home. The connecting links between the specialist and industry are already being forged, and I believe that centres for industrial rehabilitation, which will be of benefit to the whole community, will soon emerge. What will be lacking, however, if care is not taken, is close liaison between the general practitioner and the specialist. This important link was, unfortunately, weak before the war. Many general practitioners regarded acute injuries of the head as being beyond their scope and naturally tended to avoid taking responsibility for them, with the result that they were apt to be overawed by their seriousness and, consequently, had their scientific judgment coloured by impression and by the argument of anxious relatives.

The interests of an injured man must be protected, and no one can better do this than the general practitioner. It is, therefore, his duty at least to be acquainted with the basic principles of head trauma if he is not to give his important judgment merely on impression. I believe that any general practitioner will find perusal of this book not only of value but will find the subject interesting and even exciting.

Again, in this second edition I have endeavoured to make the story continuous and to avoid the temptation of overemphasising the unusual and dramatic. Moreover, I hope I have refrained from oversimplification at the expense of accuracy.

The theory of injury to the brain by the forces of rotation has been put into mathematical language by Dr Holbourn of Oxford, and this important subject is discussed in some detail in the first chapter. It is a problem in which I have long been interested and later I hope, with Dr Holbourn, to say something further on this matter.

Rehabilitation is the subject of a new chapter. It is a form of treatment that has come to stay; the principle is established;

all that has to be done now is to decide on the details My chapter on this subject deals with what rehabilitation is and how it is carried out and is not an attempt at its justification The success of the Centre at Callaly Castle has been due to the genius and sincerity of the Commandant, Mrs Leather Culley, and of the Matron, Miss Coulson. The necessary bulwark of democracy has been provided by the War Organisation of the British Red Cross and Order of St John. May I now express the opinion that when planning for the future a Centre must be under the control of some person who is "big" enough to prevent it running the danger of losing its democratic identity

As one circles around a complex subject and views it from different angles, slightly different conclusions about the same problems may be formulated If these varying conclusions are written down in their contexts certain slight inconsistencies in the whole may result. It is on these apparent inconsistencies that the professional critic is so apt to pounce without fully realising how they arose.

In the Preface to the first edition of my book I omitted to express my thanks to Dr Boyes of Edinburgh, to Dr Wyse of Salford Royal Hospital and to Dr Whitehead of the Pathological Department of Manchester University for their careful perusal and correction of the proofs I should like to thank them now

To Dr Boyes and Mrs Condon I am particularly indebted for the meticulous care which they have taken in correcting the proofs of the second edition, and I cannot say how much I appreciate all they have done.

Dr Walshe, Professor Jefferson and Mr Norman Dott have maintained and, to a large extent, set the high standards of neurological thought and neurosurgical procedure in this country, and I owe to them not only the benefits of criticism but also of precept.

To my Staff, past and present, of the Neurosurgical Centre at Newcastle-upon Tyne I should like to express my appreciation of all the care they have taken in nursing the injured back to health, and to thank them for the patient aid they have given me in making detailed observations, in particular, I should like to thank Dr Norman Whalley for his devoted and loyal assistance

Since January 1941 every fatal case of head injury which has come under my care has been subjected to post mortem examination This essential part of the study of head injuries has been made possible through the interest and co-operation of Mr Bamburgh of the Autopsy Department.

To Professor Shaw of the Faculty of Pathology in the University of Durham I am most grateful for the detailed examinations of and reports on the many pathological specimens sent to him from my Unit.

Further drawings have been made by Miss Dorothy Davison, and again the quality of these speak for themselves. I regret that my old friend and colleague now finds it so difficult to come to Newcastle.

From Dr J. A. Charles, now of the Ministry of Health, and from Dr G. P. Harlan I have received that encouragement which is necessary when engaged in other than routine work.

I should also like to thank Professor Sir Francis Fraser of the Ministry of Health for his kind permission to use E.M.S. material.

Everyone is indebted to Miss Bousfield. It is she who has participated in the hard work of collecting data and checking references; without her aid and skill in providing for me protected moments this work would not have appeared in this year.

Finally, to my friend Charles Macmillan I wish to say how much I appreciate his skill, keenness and patient understanding. His motto is "A thing of beauty is a joy for ever." What a lot publishing owes to him.

The difficulty of writing a book is being allowed to write it. If any reward were forthcoming for the efforts I have made I would ask that I be granted reasonable facilities for research work in my own Unit and for a sound-proof room of my own.

G. F. ROWBOTHAM.

FAIRHOLME,
WYLAM
1944.

PREFACE TO FIRST EDITION

THIS book has been written primarily for those who are responsible for the treatment of acute cerebral trauma and who have not received a special training in neurosurgery or in neurology It will also be of value, I believe, to senior students about to be confronted with the complexities of injuries of the head in the near future.

An effort has been made to present a continuous picture of the various problems concerned, from the moment of the infliction of violence to the stage of complete recovery or of invalidism. This is important, since what happens in the acute phases materially affects what follows, both as regards complications and sequels

In the chapter on diagnosis the temptation to oversimplify what is essentially a complex problem has been avoided, since anything but a true presentation of the facts only leads to confusion

It has, of course, in a book of this size been impossible to cover every variety of fracture of the skull and cerebral injury, but I hope a starting point has been made from which a rational approach to the subject is possible The references are not exhaustive, they were not meant to be so They do, however, give an introduction to the literature on each of the subjects considered and will lead to most of the important ones not mentioned

Most of the observations on which this book is based were made at the Stockport Infirmary, where the fullest facilities have always been granted me for clinical and research work Many of the autopsies were done in conjunction with my friend Mr Andrew McGill, and by the courtesy of Mr Ferns, the Coroner

To Dr F M R. Walshe I am sincerely grateful for reading the typescript and for the many valuable criticisms he made. I felt that if I could satisfy him I could await future judgments with confidence

I would also like to thank Mr Norman M. Dott for much useful advice and encouragement in the early stages.

The drawings have all been made by Miss D. Davison, and to her I am most grateful, not only for her skill which speaks for itself but also for her patience.

To Mr Macmillan many writers in recent times must be thankful for the high standards he has set in publications, and I appreciate also his sustained understanding.

Without Miss Bousfield's and Miss Hudleston's help this book would never have been completed.

G. F. ROWBOTHAM.

FAIRHOLME,
WYLAM
1942

CONTENTS

FOREWORD

THE medical profession has been increasingly aware of the need of an authentic treatise on head injuries, and it will assuredly welcome this volume as meeting that need admirably With modern development of mechanisation in transport and industry, accidental injuries have progressively and alarmingly increased During the past three decades orthopædic surgery has become increasingly organised and in the last few years its application to the casualties of industry and transport has been promoted and developed on a national scale. Thus an important advance has been achieved and is being developed in caring for the injured limbs of the community

What of the injured heads? Head injury has a high incidence in modern civilian casualties. Its toll in fatalities and—still worse—in serious and permanent mental and physical incapacity is heavy That this devastation can be very significantly mitigated by the combined application of neurology, psychiatry, and neurosurgery to these casualties is recognised Yet no adequate treatise on the subject had appeared in Britain until Mr Rowbotham met the challenge with this volume Still less had any widely organised effort been made to meet the special needs of the head injured

In this state of unpreparedness in this particular field the present war has descended upon us. Whereas in peace time the proportion of disability due to brain diseases is relatively high as compared with physical injuries of the brain, in war—and especially in modern "total war"—the proportion due to physical violence may be expected to be relatively much higher Thus while a clamant need for a book such as this existed before the war, it is now an acute need Britain to-day owes much to the author for meeting it. His book is a work of art, in which a sympathetic care and understanding of the injured is the strongest note, yet now it is also as a weapon in our hands by means of which a not inconsiderable part of our enemies' activities will be frustrated Meantime the advent of war has jolted the "powers that be" into organising nationally for the care of the head injured, and 'Head Injury Centres' have been established and are developing under the guidance of such men as our author With this book

to indicate the means and this organisation to put them into effect we shall better meet the stress of war; and we shall also emerge upon the peace better equipped to care for our head-injured than we were in 1939.

Why had the care of our head-injured lagged behind other medical activities? The devious ways of advance of medical science are indeed curious The specialists obviously equipped to deal with these casualties had failed to take them in hand. Neurologists and psychiatrists saw little of them, presumably because they were labelled "surgical" from the circumstance that they had acquired their disabilities by physical violence. Neurosurgeons—a small and recently established sect—were for the most part fully occupied with the surgery of brain disease, and most of them had not yet extended their activities to deal with brain injuries. Thus, in general, the head-injured did not receive attention from those specially equipped to deal with their needs, and the care of head injuries remained a Cinderella of medical science.

The author dedicates this volume to his teacher, Professor Geoffrey Jefferson. He does so with good cause. Among the many wise and beneficient activities of this outstanding figure of British medicine, the care of the head-injured has been a constant preoccupation Jefferson did good work on head injuries and made important contributions to this subject in the last war, and has continued this in his school at Manchester since. His senior pupil, our author, has not failed to realise the importance of the subject, and has so ordered his affairs that he has acquired a very large experience of it.

This extensive personal experience of all aspects of the care of head injuries, coupled with a notable capacity for clear observation, will be found to mark every page of the volume.

The author is known as a neurological surgeon. His book shows a wide grasp of the problems raised by brain injury—neurological, psychiatric and social, as well as surgical. He makes it clear that he who would treat these patients successfully must have considerable training in and knowledge of each of these sciences, and must work in close collaboration with other specialists in them. I believe that this penetrating exposition of the scope of the subject will in the future prove of even greater import than the excellent practical instruction which this book supplies and which we so urgently require at the present time.

NORMAN M. DOTT

EDINBURGH,
1942

CHAPTER I

THE MECHANISMS OF INJURIES OF THE HEAD

THAT the physics governing cranial and cerebral injuries are complicated can be readily understood, since the head is a complex body and since the violences to which it is subjected are variable in magnitude, direction and area of application

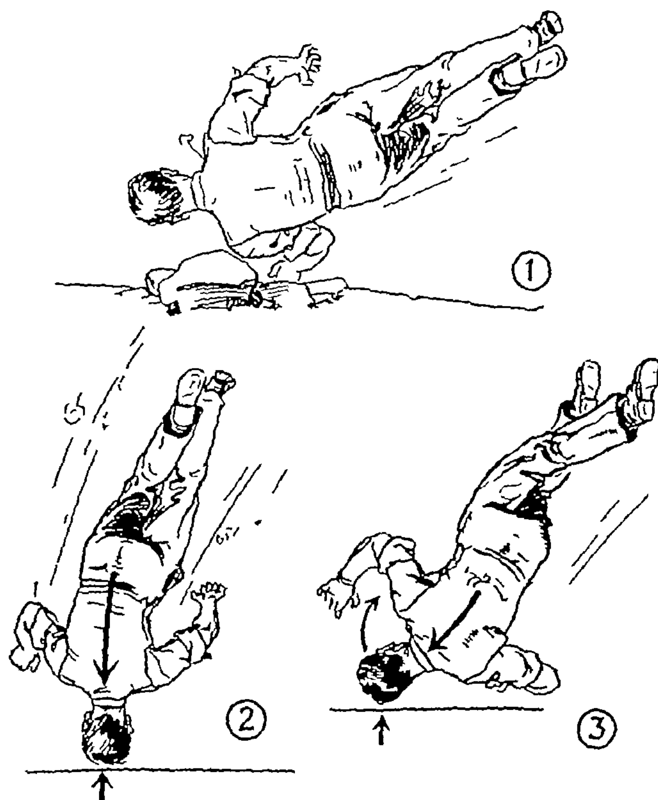
To add to the complexities, all degrees and types of injury may occur either singly to the scalp, skull or brain, or to any combination of these three structures The scalp may or may not remain intact. When broken, it may be lacerated in a simple way as when it is cut cleanly with a knife or by a piece of flying glass *Simple cuts can also occur when the head is struck with* a broad surface, because the skull is rounded and, therefore, the skin can only be subjected to shearing or cutting forces over a relatively narrow band or line When the bare head scrapes along the ground the skin is not only torn but contused and pieces may be ripped completely away Occasionally the whole scalp is avulsed. This kind of accident occurs in industry when the hair is caught up in moving machinery

The skull may not be fractured, on the other hand, it may suffer any degree of injury from a simple undisplaced linear fracture to a complete disintegration. Important intracranial structures, such as the cranial nerves, are often contused or lacerated, and fracture lines may run into the paranasal air sinuses to cause internal compounding with resulting dangers of infection Pieces of bone may be depressed which may or may not pierce the dura and lacerate the brain. In gunshot or similar types of wound the missile may be arrested by the bone without causing damage to deeper tissues, or it may pierce the bone and be arrested in the brain tissue, often it traverses the whole of the head. A core of disintegrated tissue may be the total result of the brain damage. In some cases the whole of the brain is pulped

As a result of closed injury the brain suffers chiefly diffuse neuronal damage of submicroscopical dimensions, although usually in association with macroscopical local injury of the contusional and surface hæmorrhage type

In actual practice, sufficient data to permit a precise recon struction of the accident are rarely forthcoming, but none the

less a visualisation, particularly of the ways in which injurious forces may be transmitted to the brain, is essential if the problems of cerebral trauma are to be approached logically. Fortunately the mechanisms concerned in fracture of the skull and brain damage have aroused a good deal of interest, with the result that much has been written on this subject.[1-7]



Figs 1 to 3

The Physics of Injury to the Head.

A patient may fall or may be thrown and brought to rest —

(*a*) By some part of his body, other than his head, coming into contact with a distant object—for example, the ground (No 1), or

(*b*) By his head hitting the ground—

(i) And crushed by the weight of the body when the injury force passes through the centre of gravity and the spino occipital attachment (No 2),

(ii) And set into rotation when the head is struck away from the centre line of force (No 3)

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[1] von Bergmann, E "System of Practical Surgery," i

[2] Duret, H "Traumatismes craniocerébraux" *Librairie Felix Alcan*, 2, Part I Paris, 1920

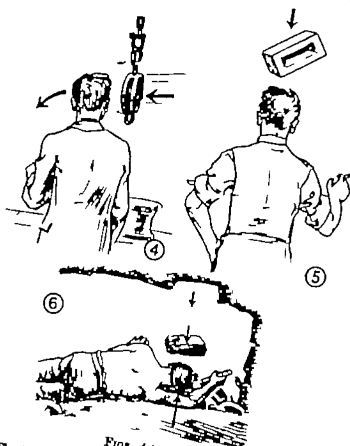
[3] Saucerotte, N "Mémoire sur les contrecoups dans les lésions de la tête" *Mém pour le Prix de l'Acad roy Chir, Paris*, 1778, **10**, 282

[4] Chopart, F "Mémoire sur les contrecoups dans les lésions de la tête" *Mem pour le Prix de l'Acad roy Chir, Paris*, 1778, **11**, 137

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[6] Polis, A *Rev Chir*, 1894, **14**, 273, 645

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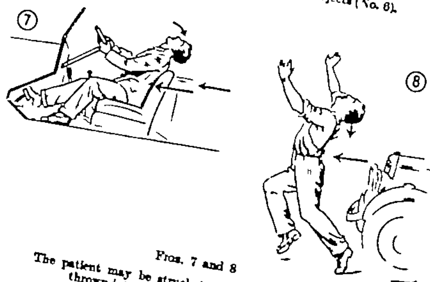
FIGS. 4 to 6

The head may be struck by a large object when the patient is stationary —

(a) And knocked away and set into rotation (No. 4); or

(b) Crushed against the spinal column (No. 5); or

(c) Crushed between two large external objects (No. 6).



FIGS. 7 and 8

The patient may be struck in the back and his head thrown into violent rotation (Nos. 7 and 8).

FRACTURES OF THE SKULL

Since bone possesses the property of elasticity, it will bend whenever a force of sufficient magnitude is applied to it under the right conditions, but whether it breaks or not depends on the degree of bending to which it is subjected. Bending certainly is the means by which most fractures of the skull are produced, and the injuring force acts either by deforming a circumscribed area of bone or by distorting the whole skull. The exact manner in which the bone breaks is determined by the fact that its tensile strength is less than its power to resist compression. Therefore, whichever table of the skull happens to be on the convexity of a bend, and thus subjected to stretch, will be the one to fracture first. This sequence of events in the solution of osseous continuity is well illustrated in the snapping of a stick across the knee.

However, before the skull can be locally deformed or generally distorted, and thereby fractured, it must offer some kind of resistance to the violence which is applied to it; otherwise the head would merely be moved through space without undergoing structural change. In other words, a counter force must act on the skull at the same time as the injuring force if a fracture is to occur. When the head is crushed between two external objects, the points of action and reaction, as the sites of application of the injuring and reactionary forces may be called, are obvious. Crushing in this way, however, happens to be a very rare type of accident. In the majority of cases the point of reaction must be in the region of the craniospinal junction, as this is the only point at which the head is tethered and resistance possible. This leads us to a consideration of the mode of attachment of the head to the body. The occipito-atlantal and the atlanto-axial articulations are so constructed that the movements of the head in relation to the spinal column have the same freedom as a ball-and-socket joint. Therefore, within a limited range, the head can move away from an injuring force and tend to escape damage, but not fully, since a certain amount of force is always expended when a body is put into motion from a position of rest or when its motion is increased.

Movements beyond the range of the occipito-atlanto-axial joints are prevented by the strong relatively inelastic occipito-atlanto-axial ligaments, and these structures also resist vertically or longitudinally directed forces which tend to lift the skull from, or drive it across, the spinal column. Thus the tension of the craniospinal ligaments sometimes acts as a counter to the injuring force.

In ordinary circumstances of accident, forces tending to knock the head off the spinal column are rare, whereas forces directed downwards and tending to crush the head against the spinal column are extremely common Thus the rigid support which the skull receives at its occipital condyles is one of the most important anatomical features concerned in the mechanisms of fracture of the skull and injury to the brain



FIG. 9

A typical indentation due to local violence. The inner table is fractured at the apex and the outer table at the periphery

Fractures due to Local Deformation.—The many factors concerned in local deformations of the skull are most obvious when definite indentations occur A small mass striking the head squarely will, if travelling at a sufficiently great speed, drive inwards *a piece of bone shaped as a cone-like indentation* (Fig 9). At the apex of such a cone the inner table will be stretched, whereas the outer table will be compressed, and thus it is the inner table which fractures first. Fracture of the outer table follows as the force continues to act, and the completed fracture line or lines must run from the central point radially Occasionally fractures restricted to the inner table, unless revealed by

radiography, pass unrecognised at the time of the injury, although later they may give rise to traumatic epilepsy if a spicule of bone has happened to pierce the dura. Sparing of the outer table in these cases is explained by the mechanics of the fracture rather than by any particular brittleness of the inner table itself

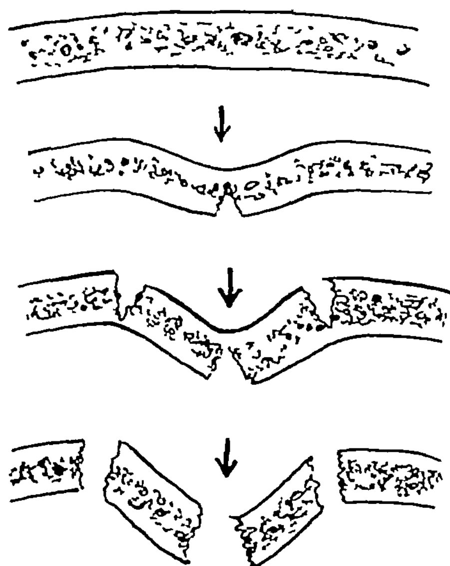


Fig 10
The sequence of events in fracture by local deformity

At the periphery of the indentation the bone is bent in the opposite direction, the convexity of the bend being outwards, and here the outer table fractures first; also the fracture lines produced tend to run circularly to enclose the base of the indentation. When the injuring force has not been expended after it has produced a complete circular fracture, a piece of bone fragmented by the radial fracture lines will be loosened and then depressed to form a typical comminuted depressed fracture (Fig. 10). Massive depressed fractures are caused by large objects travelling at great speed and they are usually overwhelming and fatal.

Fractures due to General Deformation.—In virtue of its shape and the physical properties of bone the skull behaves in some degree like an elastic sphere. Therefore, whenever it is compressed, for example laterally, there is a shortening in the line of pressure, while the vertical and longitudinal diameters are increased, which means that parts of the skull distant from the site of application of the injuring force are bulged and may fracture by bending.

The head, as shown above, may be compressed in one of two ways:—

(*a*) Between two external objects, such as the ground and one of the wheels of a motor car.

(*b*) Between an external object and the spinal column.

The latter method of compression is the more important, as it is the commoner one, and is best illustrated in motor-car accidents when an occupant of the car is thrown from his seat. In some phase of the accident the head strikes against a resistance, possibly the roof of the car, the windscreen or the ground and comes to rest, whereas the body, in virtue of its momentum, continues to travel onwards, with the result that the weight of the body through the spinal column is thrust against the occipital condyles (Fig. 11).

The same mode of compression occurs when the body is at rest and a heavy object, such as masonry falls on to the top of the head, driving the skull downwards on to the condyles of the atlas; this type of accident commonly happens to civilians during air raids when the building above them is blown down.

Fractures due to local deformation are commonly associated with those due to general distortion. For example, in falls on the

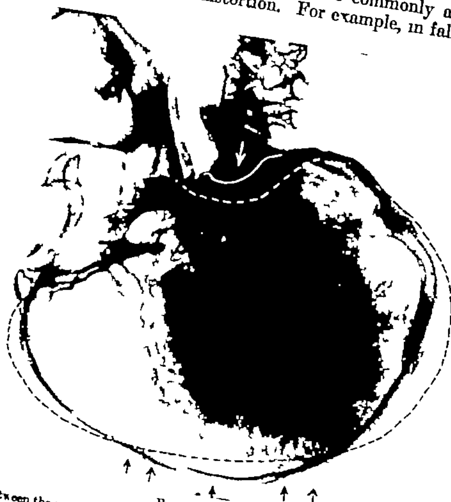


FIG 11

Crushing between the spine and an external object is the means by which fracture of the skull is usually produced. In this mechanism local, as well as general, distortion occurs.

head, apart from fracture by bursting a circular fracture around the occipital condyles, due to local deformation, may detach the basi occiput and allow it to be driven into the cranial cavity

These points were very clearly illustrated in one of my cases when a motor cyclist, travelling at speed, came into collision with a motor car and was thrown through the air a distance of fifteen yards before his head struck the ground. The impact was so great that his skull was almost completely flattened and brain tissue oozed from wide longitudinal fissures in the vault, which

were the result of bursting. Furthermore, the basi-occiput loosened by fracture due to local bending had been driven inwards and was resting against the dome of the calvarium

FRACTURE PATTERNS

As von Bruns [1] has pointed out, if the skull were equally thick and equally elastic and formed a true sphere, the lines of fracture

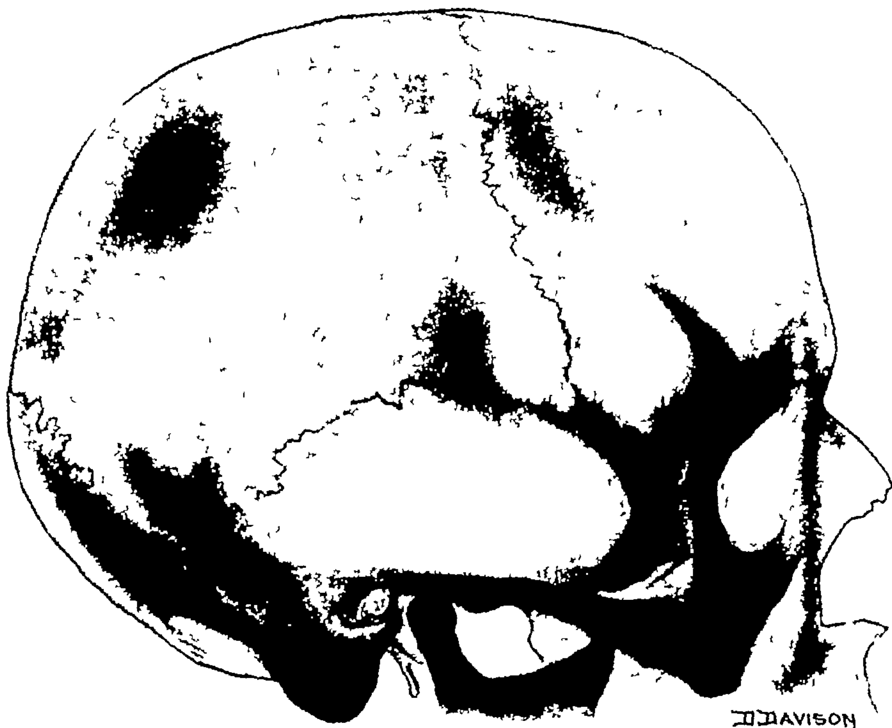


Fig 12
The weak panels and strong buttresses of the vault

could be calculated mathematically, and these would depend on the magnitude and direction of the force and on the size of the body inflicting the violence. Although the physics governing such a hypothetical system are helpful in visualising what happens in injuries of the head, important modifications have to be made for anatomical irregularities. In actual fact the skull is not a true sphere, neither is it a homogeneous body offering a uniform resistance, but is composed of relatively thin panels of bone

[1] von Bruns, P "Die Chirurgischen Krankheiten und Verletzungen des Gehirns und seiner Umhüllungen" *Handbuch der praktischen Chirurgie für Ärzte und Wundärzte* Tübingen, 1854, 1.

enclosed within strong buttresses, and this explains to some extent how fracture patterns are so numerous and why at first sight they appear subject to no rule

The Influence of Strengthening Buttresses —In the vault there are vertical thickenings at the glabella, external angular processes, mastoid bones and external occipital protuberance, and these are united by six *arches, three on each side, viz.*, the supra orbital ridge in front, the curved lines of the occiput behind and the temporal crests at the side. Also there is a stout anteroposterior arch of bone at the top of the skull in the middle line protecting the sagittal sinus The sheets of bone in the base are much thinner than those in the vault but, on the other hand they are enclosed within extremely strong buttresses One buttress runs anteroposteriorly in the middle line broken by the foramen magnum and sphenoidal air sinus The petrous bones run inwards and forwards from the sides, their weak point being external where they enclose the middle ear More anteriorly, the thickened edges of the sphenoid wings run inwards and backwards, the weak point here lying internally where the two wings separate to enclose the sphenoidal fissure The general disposition of the various bands of thickening and areas of relative thinness are shown in the accompanying drawings which were made after transillumination of the skull (Figs 12 and 13)

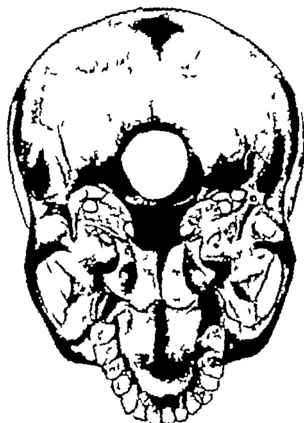


FIG. 13

The weak panels and strong buttresses of the base

In view of the above anatomical facts it can be readily understood how fracture lines often take a zigzag course, for even though a force tends to travel in the direction in which it was initiated, it also tends to follow the lines of least resistance, and

will split up into components to avoid traversing a stout barrier of bone.

Fracture patterns in the base are strongly influenced by the petrous buttress, since fracture lines which approach it from the middle or posterior fossa are turned either towards its apex or base, according to the angle at which they strike it. Only when the forces are overwhelmingly great is the middle of the body of the petrous bone ruptured, and forces of such magnitude usually cause fatal injuries. Many basal fractures converge on and



FIG 14

In this case a slanting blow lifted and rotated a piece of the side of the skull to produce a compound fracture

overrun the pituitary fossa, and this is not surprising, for not only is it central in position, and therefore necessarily often in the line of injuring forces, but it is also the point to which fracture lines will be deflected by the numerous buttresses which radiate from it.

The base of the skull, unlike the vault, is weakened by numerous foramina and fracture lines commonly open into these various openings. The sphenoidal fissure is most frequently affected, although the foramen magnum is by no means spared in spite of its thickened margins. Occasionally the whole floor of the middle fossa is loosened by a fracture which runs inwards

along the anterior margin of the petrous bone to the foramen ovale, then forwards on the side of the pituitary fossa and finally outwards through the sphenoidal fissure, to end in the region of the pterion.

The Influence of the Site of Application and Direction of the Injuring Force—A force applied to the vault and directed towards the vertex tends to lift off a dome of bone in the same way as a



FIG 15

The type of fracture produced by forces directed towards the vertex. The arrows indicate the direction of the injuring force It is of interest to note that the fracture lines run parallel with the base. Also they are widely opened and presumably act as natural decompressions, particularly as loose bony fragments are lifted and not depressed towards the brain.

knife lifts off the top of an egg, and thus extensive fractures of the calvarium may be produced which run parallel to the base This type of longitudinal fracture occurs in falls in which the head strikes a projecting object in the phase when the feet are dependent, and since the upper segment of bone is lifted away from the brain and not driven towards the base of the skull, cerebral injuries tend to be minimal (Figs. 15 and 16)

Slicing injuries, although not very common, are important since they are liable to produce compound fractures (Fig 14) The

classical sabre cut is being replaced by slashing wounds inflicted by aeroplane propellers, and one case has recently been described in the literature in which an instructor had part of his frontal bone and lobe removed by such an injury; he did not lose consciousness immediately and ultimately made a satisfactory recovery.[1]



FIG 16

An injuring force striking the skull vertically will first of all put the occipito-atlantal ligaments on the stretch Then, if the force is not expended, the dome of the calvarium, lying above the site of impact, will tend to be lifted from the base Fractures produced in this way are circular in the transverse plane Often they are best demonstrated by an oblique X-ray view as shown above

In one of my own cases a youth received a similar injury when attempting to change the tyre of a motor lorry. The metal band clamping the tyre in position flew loose as he levered it away from the rim of the wheel and struck him in the orbit beneath the supra-orbital ridge and sliced off the front of his head (Fig. 17).

Blows on the chin occasionally fracture the glenoid fossa, but it is rare for the mandibular condyle to be driven into the cranial cavity, partly because the head rides the blow, and chiefly

[1] D'Arcy, T N "A Case of Severe Head Injury" *Jour. R. Nav Med Serv*, 1936, 22, 242

because closure of the jaws transfers the force to the face and prevents the mandible from moving upwards. The serious effects of blows on the superior maxilla are becoming more widely recognised, for although the central part of the bone enclosing the antrum of Highmore crushes easily and absorbs even a severe shock, its internal angular process is strong and readily transmits forces to the non resistant cribriform plate which is easily fractured. Occasionally the cribriform plate is loosened and tilted inwards,



Fig 17

Loss of bone due to a sliding injury

leading to rupture of the olfactory filaments with loss of all but trigeminal taste (salt, sour, bitter, sweet), and often to laceration of the dura, with the danger of meningitis, since the fracture opens into the nose.

Because of infection and meningitis, blows at the root of the nose are particularly dangerous as they are so prone to lead to fractures involving the paranasal air sinuses[1] When the skin of the forehead is broken such fractures are obviously compound, on the other hand, when the skin of the forehead remains intact they are none the less compound, although only in an internal sense Internal compounding is rather the more dangerous form,

[1] Schorstein, J "Compound Fronto-orbital Fractures. *Brit Jour Surg.*, 1944, 31, 221.

as it is apt to pass unnoticed and to lead to serious consequences before adequate treatment is instituted.

An oblique blow of great force applied to one side of the back of the head will start a fracture in the underlying posterior fossa which crosses the middle line to enter the middle fossa of the opposite side, possibly to end in the anterior fossa. Messerer,[1] in particular, has stressed the influence of the direction of the injuring force on the line of fracture and has enunciated a law to the effect that longitudinally or transversely directed forces always produce fractures in the corresponding axis.

Rawlings [2] was of the opinion that most basal fractures are the result of forces applied at the level of the base and which imitate a chisel action which prises open the bone in the line of the injuring force. Some certainly are produced in this way, but it is by no means the only possible mechanism, although it is an important one. Aran [3] showed that many fractures of the vault are vertical and that they are continuous with fractures in the nearest basal fossa. On this evidence was evolved, though not by Aran, the theory of irradiation which postulates that basal fractures are purely the lower continuations of fractures which originate in the vault

In fact, basal fractures may be produced in a variety of ways :—

(*a*) By forces applied directly at the level of the base.
(*b*) By general distortion of the skull wherever the forces are applied.
(*c*) By extension from the vault.
(*d*) By forces applied to the base through the spinal column or face.

There may be no external bruising, and often details of the mode of the accident are lacking. Occasionally evidence from the above sources may help in deciding by which mechanism the fracture was produced, or an opinion may be formed from the pattern of the fracture itself. For example, a ring fracture around the basi-occiput is due to the thrust of the spinal column. In severe injuries the complexity of the fracture pattern often suggests that several factors have been active and have combined to produce the extensive and comminuted fractures of the base sometimes found (Fig. 18).

Apart from anatomical irregularities and the variability of the site of application and direction of the injuring force, there are probably other factors, though these are difficult to prove,

[1] Messerer "Ueber Elasticität und Festigkeit der Menschlichen Knocken." Stuttgart, 1880
[2] Rawlings, L B "Surgery of the Skull and Brain." London, 1912
[3] Aran, F A. "Recherches sur la fracture de la base du crâne" *Arch Gén Méd*, 1844, **6**, 180, 309

which explain how fracture lines occasionally run at all angles or stop and then start again In road accidents in particular the head may be struck more than once under different conditions, the final fracture pattern being the result of more than one injury Moreover, the skull is often struck over a wide area, which means that the injuring force is applied simultaneously at many points

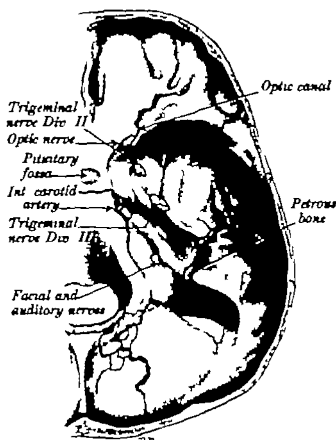


FIG. 18
Such fracture patterns are probably due to a combination of injuring forces.

that may change in position during the different phases of the injury as the skull is flattened and deformed

INJURIES OF THE BRAIN

Apart from penetrating wounds of the head the brain may be injured in three ways —

I By distortions of the skull
II By movements of the brain in relation to the skull (as, for example, when the head is thrown through space and then brought to rest)
III By acute compression of the thorax

I.—DISTORTIONS OF THE SKULL

In infants, while the sutures are still open and the bones plastic, the brain may be injured by distortion of the skull without a fracture occurring. When, however, the sutures are firmly closed and the bone has become brittle by calcification, any deformation sufficient to inflict an injury on the brain is usually of sufficient degree to produce a fracture.

From the tables in Chapter II it will be seen that the incidence of fracture of the skull in a clinical series of a thousand cases of acute head injury was as low as 68 per cent., whereas in fifty consecutive autopsies it was as high as 90 per cent. The difference of the fracture incidence in these two groups is due chiefly to the fact that fatal injuries of the brain are frequently

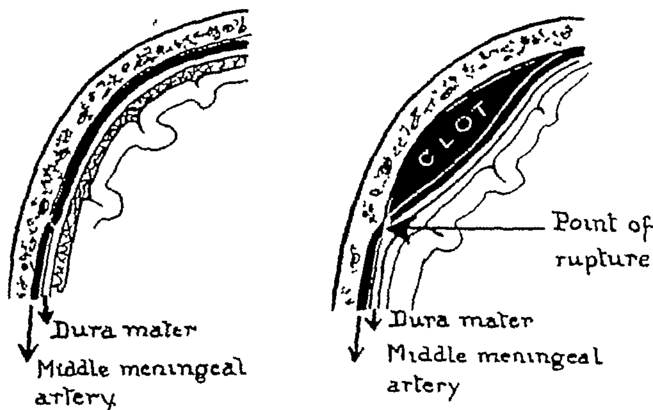


FIG 19

Rupture of the middle meningeal vessels by stretching at the point where they leave a bony tunnel or groove to become attached to the dura

caused by overwhelming violences which the cohesion of the skull cannot possibly resist.

During distortions of the skull there is a lag in the moulding of the intracranial contents to the new curvatures of the bone, so that parts of the brain are subjected either to increased pressure or to suction, and no part of the brain escapes the effect of the injurious forces. Also, as in the second mechanism, the tissues of the brain are bent, stretched or torn.

Moreover, as the skull rebounds after the injuring force has ceased to act, important structures may be torn, and, in particular, if the dura does not follow the movements of the skull the meningeal vessels will be pulled away from the bone and small nutrient vessels broken. Rupture of the main vessels themselves depends on certain anatomical peculiarities. Often the vessels are embedded in a deep bony groove, with overhanging edges in the proximal part of their course, whereas from the Sylvian point onwards they are firmly attached to the dura

and, therefore, will break at this point of junction whenever the skull moves away from the dura (Fig 19)

The severity of an injury to the brain consequent on deformity of the skull depends on the circumstances of the accident. For example, let us consider what may happen to a man when swimming round a ship at anchor If the ship rises slowly on the swell of the tide and knocks against his head in the open sea, he will merely be pushed away with very little damage having been inflicted On the other hand, if he is swimming between the ship and the quayside the same movement of the ship, if he is trapped, will crush his head to pulp

Probably, as will be shown later, cerebral injuries are more frequently caused by the forces associated with movements of the brain than through deformities of the skull, but, on the other hand, overwhelming types of injuries to the brain are often the result of forces continuing to act after the skull has been deformed and fractured

By experiment it has been shown that when the head of an animal is crushed by means of a falling weight unconsciousness does not result until the deformity of the skull is such that the intracranial pressure rises to or above that of the systolic blood pressure [1]

II.—MOVEMENTS OF THE BRAIN IN RELATION TO THE SKULL

1 Injury by Linear Movements.—When a man is thrown on his head against a resisting object the skull, at some phase of the injury, becomes stationary, whereas the brain, by virtue of its momentum, *continues to travel onwards in the same direction* and is injured by those forces which resist its movement and finally bring it to rest.

The advancing surfaces of the brain come into forceful impact with segments of the skull or with the faces or edges of the dural septa, according to the circumstances of the accident. Because of their rigidity and their firm attachments to the bone the dural septa act as parts of the skull in resisting the movements of the brain

When the brain is thrown from the base the upper halves of the outer surfaces of the hemispheres strike the vault of the skull, the upper surface of the corpus callosum strikes the free edge of the falx, and the upper surfaces of the cerebellar hemispheres are pressed against the under surface of the tentorium. In lateral movements the outer surface of one hemisphere strikes the side of the skull, whereas the inner surface of the opposite hemisphere strikes the flat surface of the falx, the brain stem

[1] Scott, W W "Physiology of Concussion." *Arch. Neur and Psych.*, 1940, 43, 270.

impinges against the edge of the tentorium and one side of the cerebellum strikes the lateral wall of the posterior fossa. When the brain is travelling towards the anterior fossa in the longitudinal axis the anterior poles strike the anterior walls of the anterior fossa, the rostrum of the corpus callosum the edge of the falx, the brain stem the basi-occiput, and the anterior surface of the cerebellar lobes the anterior walls of the posterior fossa. In movements towards the base the under surfaces of the hemispheres and cerebellum are principally affected. Therefore, according to the axis along which the brain moves, all combina-

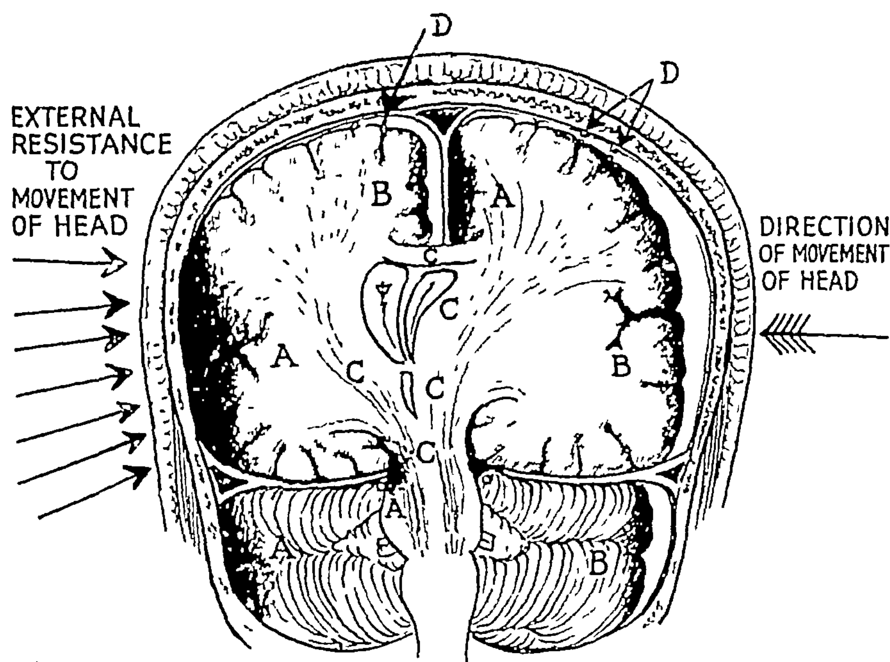


FIG 20

The mechanism of cerebral injury typical in road accidents

A, Injury by impact
B, Injury by suction
C, Injury by distortion
D, Rupture of vessels by stretching

tions of surface injuries may occur, the nature of these depending on the speed at which the brain was travelling when its motion was arrested and on the type of surface with which it made impact. For example, lacerations are particularly liable to occur when the brain is flung forcibly against the bony irregularities on the floor of the anterior fossa or against the sharp edges of the wings of the sphenoid bones.

At the pole opposite to the site of impact the brain moves away from the skull, and before the space produced can be filled with cerebrospinal fluid a zone of diminished pressure results in which suction may be sufficient not only to rupture surface vessels but also those more deeply placed (Fig. 20) The

importance of the part played by suction in cerebral trauma has recently been emphasised by Dott.[1]

Moreover, apart from changes in intracerebral pressure, the mere sliding of the brain within its dural coverings is a very serious happening for not only does it account for the rupture of tethering blood vessels but also for the avulsion of cranial nerves The vessels which drain the cortical veins into the large venous sinuses have very fragile walls, and although they often run for half an inch or more in contact with the under surface of the dura, they are firmly attached to it, furthermore, their course across the

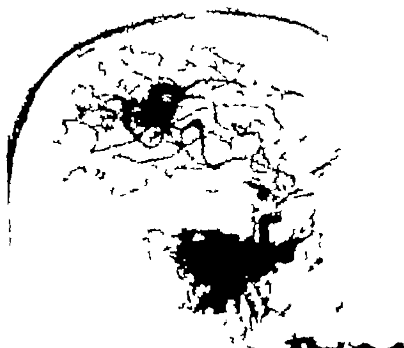


FIG. 21

An angiogram depicting the vessels of the brain, showing how a wide and safe range of movements is afforded to large cerebral vessels by their tortuosity

subdural and subarachnoid space is short and straight, and hence *they are easily torn when the cortex is displaced* It is rupture of these communicating veins which accounts for most cases of profuse subdural or subarachnoid hæmorrhages

On the other hand the large arteries at the base of the brain run a tortuous course, and a good deal of slack has to be taken in before they become taut. The tortuosity of the arteries and strength of their walls explain why they are so rarely torn (Fig 21) When rupture does occur it leads to rapid death, except in the rare instances when the internal carotid artery bleeds into the cavernous sinus, with a resulting pulsating exophthalmos The smaller

[1] Dott N M "Thompson and Miles Manual of Surgery" 1939 2.

arteries which enter the base of the brain through the perforated spots or supply the optic chiasma are much more easily avulsed.

That the whole brain behaves as a homogeneous body of uniform consistency is a very erroneous impression. It is, in fact, composed of tissues of different specific gravity, toughness and tensile strengths. Moreover, it does not act as a single unit. Each hemisphere is separate from the other, apart from its relatively narrow connections through the corpus callosum and commissures, and both hemispheres are connected with the cerebellum only through narrow pathways of the brain stem. This means that large anatomical units of the brain can move in relation to each other, in addition to the whole brain moving in relation to the skull, with the result that connecting pathways may easily be bent, stretched or torn whenever the brain is made to alter its shape. To a lesser extent the forces of distortion affect the connecting tissues between the white and grey matter and probably between every individual cell.

The brain can move within the skull in spite of the fact that its tissues cannot be compressed into a smaller volume, because it does not completely fill the cranial cavity and because the cerebrospinal fluid which occupies the extra space can be displaced from one chamber of the skull to another or into the spinal theca, to accommodate cerebral displacements

The cerebrospinal fluid also acts as a cushion to the brain and, in the mechanism of injury under consideration in this section, tends to minimise the injurious effects of cerebral movement, and particularly protects the advancing surfaces from laceration.

How much compression of the blood vessels at the site of impact leads to displacement of blood into extracerebral vessels is not known, probably not to a considerable degree, though sufficiently to cause some anæmia in the part concerned.

According to the circumstances of accident, injury to the brain by the mechanism of movement may be brought about by forces resulting either from deceleration or acceleration.

Let us suppose that a man is standing on the road when he is struck by a car. In a fraction of a second he is set into motion from zero to, say, thirty miles an hour or, in other words, he is struck by a force which causes tremendous acceleration. If a soft part of the body is struck it will give a little, thus reducing the rate of immediate acceleration and minimising the effect of the blow. If, however, the head is struck the resistant bone of the skull will not give in the same way as the soft tissues of the body, with the result that the rate of acceleration will be greater. In this case the head may be compressed against the spinal column and thus the brain may also be injured by the forces of skull deforma-

tion If a man is carried forwards on the radiator of a car which gradually comes to rest and he is not thrown, then the forces due to acceleration only are concerned in the infliction of the injury If, however, he is thrown forwards he may be brought to rest abruptly by his head striking an object which resists his motion, and supposing he had been struck over a soft part of the body he may be brought to rest in a shorter time and over a shorter distance than he was set into motion In this case the forces of deceleration may be greater than those of acceleration if the body has not lost considerable speed when the last impact occurs.

For a long time racing motor cyclists have realised how important it is to hold on to their bicycles when a crash comes so as to guard against the possibility of being thrown on to their heads and thus being injured by the forces of deceleration, They know that after most collisions a bicycle will skid and come to rest relatively slowly

2. Injury by Rotation —The most recent studies on the mechanisms of injury to the head have been carried out by Holbourn in the Physics Department of Oxford University[1] In the opening sentence of his first publication he makes the clear statement that the behaviour of the skull and brain during and immediately after an injuring blow is determined by the physical properties of the skull and brain and by Newton's laws of motion

Given, therefore, a knowledge of the primary laws of physics, it is necessary to consider the physical properties of the skull brain system the important ones being —

(a) The comparatively uniform density of the brain and interstitial tissues, both being approximately the same as cerebrospinal fluid

(b) The extreme incompressibility of the brain tissue—a force of 10,000 tons being necessary to compress the brain to half its volume. By compression is meant forces acting equally in all directions so that the cubic capacity of the tissue is lessened but its shape unaltered

(c) The small modulus of rigidity of brain tissue, which means that the brain is very easily pushed out of shape

(d) The great rigidity of the skull—a force of 1 ton being necessary to reduce the diameter of the skull by 1 cm

(e) The shape of the skull and of the enclosed brain

It is reasonable to assume that, as in the substances used in engineering, brain tissue is damaged when its constituent particles are pulled so far apart by the injuring force that they do not come together again and unite properly when the blow is over If this is what really happens, then because of the properties of the

[1] Holbourn, A. H. S. "Mechanics of Head Injuries." *Lancet* Oct 1943, 145 438.

skull-brain system, brain damage must be due chiefly to shear strain and not to the strains of compression and of rarefaction or suction. By shear strain is meant the slide of one part of a tissue across another, as is seen in the wobbling of a jelly.

Distortion of the skull certainly alters the shape of the brain, although, according to Holbourn, in view of the physics of the skull-brain system this can rarely be of sufficient degree to cause serious widespread damage in the brain tissue. Since distortion is always greatest at the site of application of a blow, generalised distortions of the skull are apt to behave merely as local indentations and to inflict on the brain local contusional types of injury, including torn vessels and not diffuse neuronal damage, which gives rise to concussion. This probability is supported by those cases described by Russell of severe crushing of the head between railway buffers; causing severe fracturing of the skull without loss of consciousness.

Injurious forces consequent upon movement can, in the main, be divided into (*a*) changes of velocity in a straight line—linear acceleration, and (*b*) changes of velocity as the brain rotates around one of an infinite number of axes—rotation acceleration.

According to the theory of Holbourn, damage to the brain in the large majority of cases of accident must be caused by rotation which arouses severe shear strains. Given the precise details of how the head is struck, brain damage can be predicted with reasonable mathematical accuracy.

Let us, for example, consider what happens when a man falls backwards and strikes his head heavily on the ground. First, the skull takes the weight of the blow and rotates forwards on a transverse axis. Then the brain itself begins to rotate in the same direction and on the same axis, but only as a consequence of the forces transmitted to it by the movement of the skull. Therefore, the regions of the brain in which the greatest shear strains develop are those where the skull can get a firm grip on the brain and make it rotate. This applies particularly to any projecting ridge. For example, the overhang of the sphenoidal wing will stick into the pole of the temporal lobe and drag it along with its rotatory movements.

In the vault of the skull there are no projecting ridges and as a result the brain slips backwards in relation to the skull at the pia-arachno-dural interface. Vessels crossing this face are stretched and often torn. Rubbing also occurs, and by friction alone the brain is, to some extent, dragged along. Deeper tissues of the brain slide back on the more superficial ones, as shown in Fig. 22, but as the superficial layers are subjected to the first and greatest strain, it is here that the maximum injuries of this region

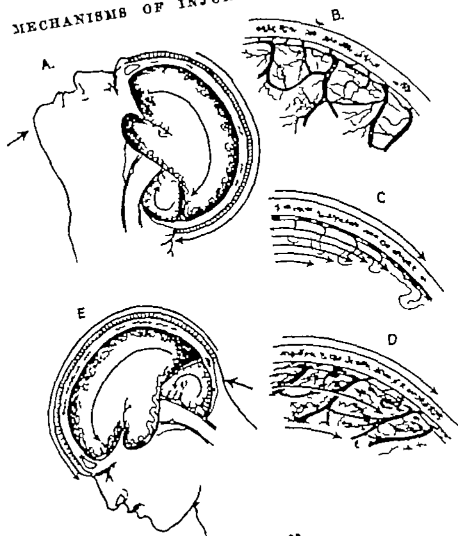


FIG 22

Injury of the brain by rotation

Whenever the head is struck by a force not directed along that line which passes through the centre of gravity of the head and the occipito-atlantal joint (the fulcrum), it is set into rotation. The skull necessarily takes the first impact of the blow and moves before the brain. Then the brain is secondarily set into motion by the skull and particularly by projecting bony prominences and dural septa. Since the brain is soft and not rigid, it rapidly becomes deformed. In closed injuries it is the shearing forces associated with deformity that cause the maximum damage to the cerebral tissues and tear the cerebral arteries and veins.

A, When a patient is struck on the chin the head is thrown backwards.

B, C, D } Showing the resulting deformity of the brain in relation to the vault of the skull.

E, If a patient falls backwards and strikes his head on the ground the head will be knocked forwards into an anterior rotation, the shearing forces in this case being in the opposite direction. Probably in most accidents, the head is set into violent rotation about different axes at different phases of the infliction of the violence.

are inflicted. Strains, other things being equal, are always greater at the junctions of tissues of different densities, and this explains why contusional injuries in the nature of petechial hæmorrhages commonly occur in the region of the walls of the ventricles.

When the head moves from side to side on an anteroposterior axis, the pituitary fossa becomes an important projecting buttress and considerable forces are transmitted through it to the brain. This means that great shear strains are developed in this region and account for many of the injuries which are inflicted on the hypothalamus or floor of the third ventricle.

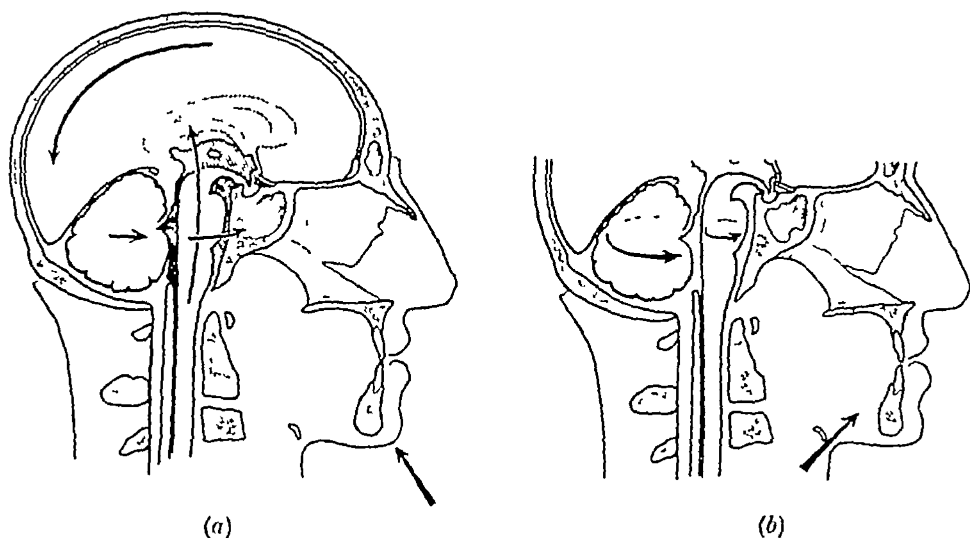


FIG 23

Rotation of the brain can occur about any axis In (a) the brain stem is subjected to stretch, in (b) to twist

Rotation, of course, can occur on an infinite number of axes and the physics will vary accordingly.

The hemispheres of the brain spin inside the vault and the cerebellar lobes within the confines of the posterior fossa. Since the cerebellum is very much smaller and lighter than the cerebrum, it is more easily set into motion and therefore much less liable to damage than is the heavier cerebrum. It has not yet been decided to what extent the brain stem is subjected to the special strains of rotation although presumably they are often those of stretch.

In order to prevent confusion it must be realised that when Holbourn uses the term "compression" he is speaking of the direct action of this force on nervous tissue proper and not on the brain as a whole. The brain consists not only of neurones but also of a vascular system composed of arteries, capillaries and veins. Even a relatively small compressional force will lead to occlusion of a large cerebral vessel and to ischæmia. Complete ischæmia, even of a few minutes' duration, will lead to death or permanent loss of function in the area of the brain concerned.

3 Experimental Injury by Acceleration —Denny Brown and Russell[1] have proved by experiments on dogs, cats and monkeys that the forces of acceleration alone can inflict on the brain the type of injury which causes concussion. By means of a specially constructed pendulum, carrying at its free end a broad striking piece made of brass, the heads of animals were struck and set into motion from the resting position. The blow was usually arranged to fall on the occipito parietal region, as here the skull

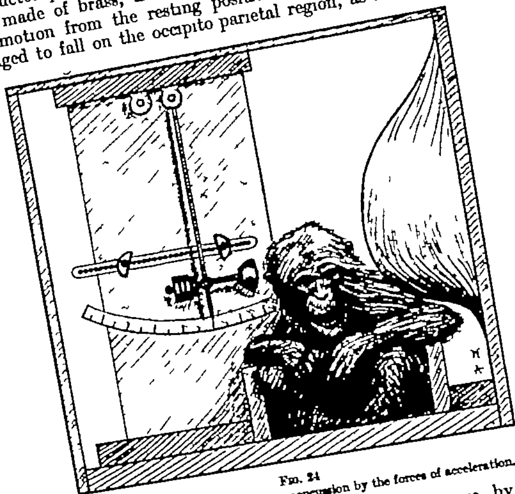


Fig. 24
Experimental method of producing concussion by the forces of acceleration.

is thickest and least liable to fracture Furthermore, by means of a stop the motion of the pendulum was arrested after having travelled 2 cm, thus preventing the possibility of crushing Also the forces of deceleration were eliminated by bringing the head relatively slowly to rest against a soft wool cushion. In all cases anæsthetised animals or decerebrate preparations were used (Fig 24)

It was found that before concussional effects could be obtained a certain degree of intensity of blow was necessary or, in other words, an adequate stimulus was required In cats and monkeys the head had to be set into motion in a small fraction of a second

[1] Denny Brown, D. and Russell, W R — Experimental Cerebral Concussion. *Brain* 1941 64, 93.

from zero to a speed of 28·4 feet per second before any appreciable effects on the brain stem, such as changes in blood pressure, respiration and corneal and pinna reflexes, could be measured. The effects produced by this type of stimulus were referred to as acceleration concussion. By direct measurement it was proved that a rise of intracranial pressure did occur when the head was struck, but was never sufficiently high to account for the changes in cerebral function the experiments produced. By microscopy the authors showed that their so-called acceleration concussion was due to injuries of submicroscopical dimensions. Furthermore, they found that in these submicroscopical injuries the brain did not obey the "all or none" law, since injuries of increasing severity would render the state of concussion more prolonged. This was a most important observation and tends, in my opinion, to show that concussion is not due to a specific type of injury to a specific part of the brain.

When a blow to the head is not of sufficient severity to cause concussion it may affect the cardiac, vasomotor and respiratory functions, through the vago-glossopharyngeal system at the base of the skull, and give rise to a state akin to primary shock. Such states occur in boxers when they receive a knock-out blow on the chin.

When a blow is above the threshold value necessary to cause concussion, injuries of macroscopical dimensions such as contusions and lacerations may be inflicted on the brain tissue This means that in nearly all cases of severe head injuries there are imprinted on the general background of concussion the various clinical pictures due to contusional types of injury. In particular, Denny Brown and Russell found that petechial hæmorrhages were specially liable to occur just beneath the pia mater in the upper segments of the spinal cord. This is another important observation, since severe cervical spinal cord injury is by no means an uncommon complication in cerebral trauma and may be the primary cause of death or disability. Because of the rapidity by which the small hæmorrhages occurred, the authors came to the conclusion that they were caused by direct rupture of the vessel as a result of squashing or stretching and not by diapedesis. By diapedesis is meant damage to the endothelial lining which allows the blood corpuscles to seep through the vessel's wall into the surrounding tissues.

Since acceleration concussion is dependent on the rate of increase in velocity, it will be seen that anything which damps down the blow will materially protect the brain. This, of course, is the principle of the crash helmet which is proving so valuable in the prevention of head injuries amongst motor cyclists in H M. Forces.[1]

[1] Cairns, H "Head Injuries in Motor Cyclists" *Brit Med Jour*, 1941, 2, 465

4 **Injury by Contrecoup** —It has often been stated that damage to the brain can occur by "contrecoup," but what is meant by *this mode of injury has never been made very clear* Usually the term "injury by contrecoup" is used purely in the sense that a pole of the brain opposite to the site of impact has been damaged and does not indicate the particular physics by which the injury was produced (Fig 25)

This limited implication of the term is probably the correct

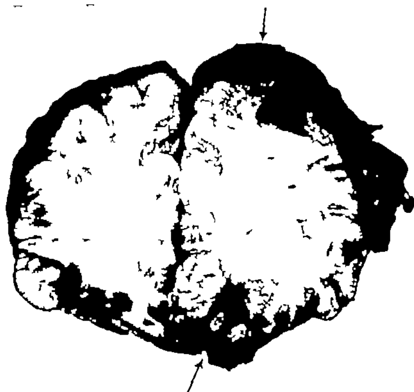


Fig. 25
A good example of contrecoup injury of the brain.

one, as cerebral lesions of a contrecoup distribution can readily be explained by one or other of the above mechanisms, and they are produced in four ways —

(*a*) By the shearing forces of rotation

(*b*) By suction, either when the brain is flung from the skull when the motion of the head is abruptly *arrested against* a resistance or in distortions of the skull.

(*c*) By the brain being struck by a distant arc of the skull as it is flattened in a deformation

(*d*) By the brain being thrust against a dural septum or face of the skull when its opposite side is struck by a local inbending of bone—linear acceleration

III.—ACUTE COMPRESSION OF THE THORAX

Stewart, Russell and Cone,[1] basing their opinion on the correlation of the clinical signs and post-mortem findings on a stunned





FIG 26

Following acute compressions of the thorax, petechial hæmorrhages not only occur into the skin of the face and neck but also into the brain tissue itself

pheasant which was found at the edge of a bomb crater, came to the conclusion that a hæmorrhage in the brain had resulted from acute thoracic compression

[1] Stewart, O W, Russell, C K, and Cone, W V "Injury to Central Nervous System by Blast Observations on a Pheasant" *Lancet*, 1941, **240**, 172

Ascroft[1] describes the case of a soldier who suffered from a typical blast injury of the lungs without evidence of gross cerebral damage At autopsy an extensive lesion of the brain was found, consisting essentially of a large number of small hæmorrhages in the grey matter of the cortex

A third case of a thoracic injury leading to probable cerebral damage was discussed in 1943 at a Staff Meeting of St Hugh's Military Hospital, Oxford A man had received a "run over" injury to the chest without head injury and, according to neurological and psychiatrical opinion, he exhibited those symptoms and signs which commonly follow organic injury to the brain

TYPES OF BRAIN INJURY

1 **Lacerations** —Lacerations occur at those sites where injurious forces develop their greatest magnitude, during movements of the brain such a concentration of force occurs in the region of bony buttresses and irregularities, and about sharp dural projections They also occur in the region of local deformities of the skull and at the points of impact in general deformities In local deformations a piece of skull may be driven into the brain and act as a cutting edge when shearing forces of great magnitude develop Without the tough covering of the pia mater, laceration of the brain would be much more frequent. Tears of the dura mater, dural sinuses and meningeal vessels must be due to deformities of the skull and almost invariably result from fractures They cannot result from movements of the brain, because the dura is firmly attached to the bone and moves with it. In other words they are essentially features of skull fractures.

2 **Contusions** —Contusions are caused by the same physical agents as lacerations Moreover, as they may be inflicted by forces of lesser degree, they occur in association with, but beyond the periphery of, lacerations. Also, as stated before, they are found at the junctions of tissues of different densities where shearing forces are large It is unlikely that they are ever caused by the percussion of a rapidly moving wave of cerebrospinal fluid

3 **Diffuse Neuronal Injuries.**—Whatever may be the exact means by which a brain is injured, and whatever the extent and severity of lacerational and contusional damage, the physics of closed injuries to the head are such that the whole brain is subjected to the injurious forces in some degree or other

The exact nature of the neuronal damage is not known. It seems, however, justifiable to enunciate the following hypothesis Probably the internal structure of a large number of cells is altered

[1] Ascroft P B. "Fatal Case of Blast Injury of Lungs. *Jour R A M C.*, 1943, 80, 142.

in the same way as a unit of tissue is altered following stress. The constituent particles take up different positions in relation to each other within the cell membrane. This no doubt gives rise to complex chemical and electrical changes—changes which lead to disorder or paralysis of function, disordered function being probably as important as paralysis.

The synaptic junctions between contiguous cells are also probably damaged, so that neural messages are not conveyed from one unit of the brain to another. In this way, even though each individual cell maintains its normal function, the integration of the whole function of the brain is changed and impaired.

The brain does not act as isolated units but as a complex system of co-ordination.[1] For example, skilled movements of the fingers amongst other things are dependent on accurate sensory messages, general balance and adequate fixation of the shoulder, elbow and wrist joints.

Neural damage of submicroscopical dimensions has long been suspected as being the essential cause of concussion, and that such injury can affect the normal functioning of nervous tissues has been adequately demonstrated by the experiments of Denny Brown and Russell.

Injurious forces probably not only affect brain functions through direct action on the nerve cells themselves, but also indirectly by interfering with their nutrition through their blood supply. It is known that a large artery can be set into complete and prolonged spasm by an injury to its coat, and probably severe ischæmia can also be brought about by paralysis of vasomotor mechanisms as a result of a general shake-up of the brain. Cases are now being published of infarction and wastage of large areas of the brain due to late thrombosis following injury to the endothelial lining of large vessels.[2]

Secondary compressional forces of great importance may develop as the result of venous congestion, extravasation of blood and of œdema. More, however, will be said on these subjects in the next chapter.

4. Penetrating Wounds in Peace Time.—In peace time penetrating wounds of the brain are rare, because the circumstances of the accident are such that a small mass does not often strike the skull at a sufficiently great speed to pierce the thick and tough bones of the vault. When they do occur they are usually due to indriven fragments of bone, or less frequently to pointed metal instruments or to such things as the spikes of iron railings. The

[1] Hughlings Jackson, J "Selected Writings of John Hughlings Jackson," vols i and ii. Hodder & Stoughton London, 1931

[2] Cairns, H Paper read at Meeting of Neurosurgical Society, London, July 1942

type of force which causes a depressed fracture of the vault usually bursts the scalp and comminutes the bone, but even in those cases where the dura is pierced it is very rare for a bony fragment to become embedded deeply in the cerebral tissue, and rarer still for it to become completely buried

The relatively thin panels of bone at the base of the skull for obvious reasons are not vulnerable to piercing types of injury

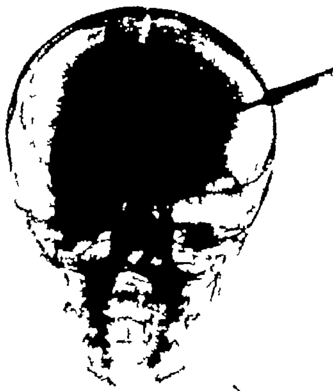


FIG. 27
A dart piercing the skull and puncturing the brain.

save at the roof of the orbit, where a penetrating wound may involve the brain, but I have known this to happen only in few instances

In one case a child fell from a tree on to a pointed stick which travelled along the inner wall of the orbit without damaging the eyeball and penetrated the frontal lobe of the brain by passing through the floor of the anterior fossa. An uninterrupted recovery was made after the stick had been removed The second case happened *in an air raid, when a piece of wood,* blown into the orbit by the explosion of a bomb, ruptured the globe of the eye

and became embedded in the sphenoidal fissure, causing a fatal meningitis. Examples have been encountered of people poking a way into the brain through the orbit or nose in an attempt to get relief from severe and intractable headaches. In one case I know of, this led to the formation of a frontal abscess.

In operations at the roof of the nose for ethmoidal sinusitis, adenoids or nasal polypi, cerebral structures may be damaged when a pre-existing intracranial lesion has led to destruction of the base of the skull. For example, I have known of a saccular aneurysm of the internal carotid artery being ruptured, a pharyngeal meningocele being shaved off and the dilated third ventricle of a hydrocephalus being opened. In each case, with appropriate treatment, the patient recovered.

5. Air-raid Casualties.—In air-raid casualties all degrees of lacerations of the scalp are exceedingly common, whereas compound depressed fractures of the skull and, in particular, penetrating wounds of the brain are relatively rare. No doubt this is due to the way the injury is produced, for a bomb fragment is usually a large piece of metal, and its initial velocity is very great, so that if it strikes the head squarely it is likely to disintegrate it completely and cause instantaneous death. Non-fatal penetrating wounds are due to small missiles, and often more than one fragment of metal is found deeply embedded and scattered in the cerebral tissue.

As far as can be judged, most cranial and cerebral injuries which occur in air raids are due to the indirect effect of blast. Either the patient is thrown on his head or his head is struck by falling masonry or by moving objects. This means that the head is injured by forces of relatively low velocity such as cause concussion. It is those of high velocity with great kinetic energy and low momentum which tend to penetrate the skull. A sufficiently heavy object, of course, even if travelling at a low speed, will completely crush the head and burst the scalp, but again this type of injury is fatal.

The direct effect of blast on the head is not known with any certainty, but from the power that it may exert, up to many hundreds of tons per square inch, it is obvious that all degrees of cerebral and cranial injury are possible. On occasions the head is blown to bits.

The effect on the brain of acute compressions on the lungs has already been mentioned. A special circumstance in air-raid damage is flying glass which is apt to lacerate the scalp and damage the eyes.

Gunshot Wounds.—Gunshot wounds of the head have aroused

interest from the time small firearms were introduced many centuries ago, and it was not surprising to find in the literature that extensive researches were made on this subject by the Imperial Royal Prussian War Department,[1] both on the living and on the cadaver At the beginning of the last war, what was virtually the textbook of military service was written by Sir George Makin[2] on his "Surgical Experiences in South Africa," and it makes extremely interesting reading When in 1922 the history of the medical services of the 1914-18 war was published, the subject of gunshot wounds of the head was reviewed by Wagstaffe[3] Already new and important contributions have been written in this war

The varieties of gunshot wounds of the head are legion All degrees of injury from metallic staining of the outer table of the skull to complete shattering of the head have been described, and Cushing[4] classified the wounds according to their depth The type, extent and depth of a wound depend on the site at which the skull is struck, on the angle of striking on the physical properties of the skull, on the speed of the bullet at the time of impact and on whether or not the bullet changes its shape after impact

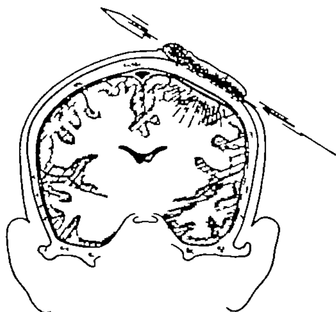


Fig 28

Local percussional violence

Exemplified by tangential bullet wound of scalp and skull. The skull is locally and momentarily displaced by the blow The bending in is of small extent but at high velocity The underlying brain is locally percussed and its function temporarily deranged. (*Norman Dott.*)

According to Wagstaffe a bullet which strikes the head tangentially may cause a disturbance in the brain sufficient to produce concussion without fracturing the skull or lacerating the scalp, but this type of injury is rare (Fig 28) Usually a bullet

[1] Coler A. G E. von, Schjerning, O von "Ueber die Wirkung und die kriegschirurgische Bedeutung der neuen Handfeuerwaffen." *Int Congr Med., XI Rome*, 1894 95 **4**, Chir 212

[2] Makin, Sir George "Surgical Experiences in South Africa "

[3] Wagstaffe W W "Official History of the War " 1922 *Med Serv Surg of the War* **2**, chapter 1.

[4] Cushing, H. "Study of a series of wounds involving the brain and its enveloping structures." *Brit Jour Surg* 1917 18, **5**, 558.

or piece of shrapnel strikes the head more or less squarely and causes a penetrating wound which, of course, is necessarily compound (Fig. 29).

Individual and racial differences in the thickness and brittleness of skulls are considerable and, when the injuring force is not of overwhelming magnitude, they may determine whether a serious or a simple injury results. For example, thin skulls of

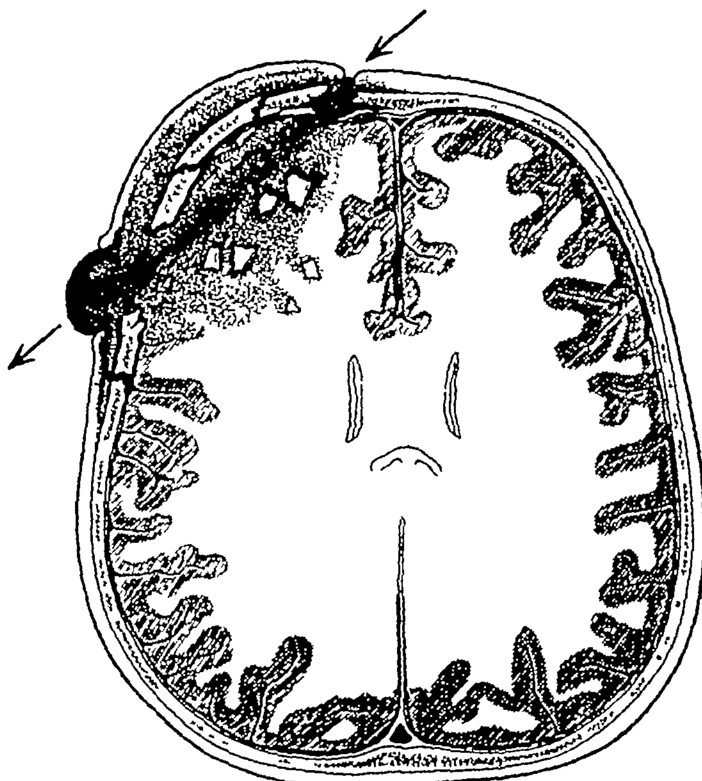


Fig 29

Violence by high velocity penetrating missile

A missile has passed through head as indicated It has produced penetrating fracture of skull at entrance and exit and has scattered fragments of bone along and around its track in brain A bursting fracture of skull has been produced by explosive effect of velocity A considerable volume of brain tissue has been disintegrated around track through brain Hæmorrhage is causing accumulation of clots and extrusion of damaged brain matter through external wounds (*Norman Dott*)

children may be pierced by a bullet at the end of its flight when it is coming to rest, whereas the skulls in some types of negro are so thick and tough that they have been known to hold up a bullet fired at point-blank range

Herodotus, while walking across the ancient battlefields at the mouth of the Nile, discovered that the skulls of the Egyptians were thick and tough, whereas those of the Persians were so thin and brittle that they could be broken by striking them with a

pebble These differences he attributed to differences in custom The Egyptians from early childhood had their heads shaved and so, by action of the sun, their skulls became thickened. On the other hand, the Persians protected their heads with turbans and so their skulls remained thinner (Figs 30 and 31)

Dumdum or soft nosed bullets which easily flatten on impact, lacerate the tissues much more severely than do "full jacketed" or hard nosed bullets which do not lose their shape and



FIG. 30



FIG 31

Differences in the thickness of skulls, however can vary considerably even in people of the same race and social customs. This difference is clearly demonstrated in the above figures.

when fired from a short distance have almost the same result as a local explosion, their shattering effect is so great.

At any point on its trajectory the force a bullet possesses depends on its momentum, which is the product of its mass and velocity Therefore, as the mass of the bullet is a constant, and as the bullet gradually loses speed from the moment it leaves the gun, the damage it is able to inflict on the head, all other factors being equal, must be proportional to the distance from which it was fired. Experiments made on the cadaver by German investigators, with a rifle firing a hard cased bullet at a velocity of 2 000 ft. per sec., showed that a bullet readily passed through the head and did not become arrested in the brain tissue until a

distance of 8,700 ft. was reached. The amount of damage a modern rifle bullet is capable of doing can be judged from its ability to pierce a brick wall 21 in. thick at battle range

When a bullet traverses the cranial cavity it not only destroys the structures in its pathway but also produces an expansile or



Fig 32

A large piece of metal lies at the bottom of the wound track, whereas smaller metallic and bony fragments (*a*, *b*, *c*) are deposited superficially

explosive effect, the forces of which are transmitted throughout the brain tissue and cerebrospinal fluid. The explosive effects of a bullet fired from a short distance may be sufficient to burst the scalp, shatter the skull and dislodge the brain. Occasionally the whole or mangled brain is blown a distance of many yards, and this happening is called “exenteratio cranii.” In the less severe types of injury a cone of damaged cerebral tissue is all

that surrounds the wound track, and between the two extremes all degrees of cerebral and cranial injury may happen

A point of interest, as well as one which illustrates the mechanism of bending in producing a fracture, is that the inner table at the entrance hole of a bullet wound is always much more extensively comminuted than is the outer table, whereas the opposite happens at the exit hole.

Of the severe types of injury the one which concerns the surgeon particularly is that in which the bullet remains in the cranial cavity In such cases pieces of bone and occasionally pieces of hair or cloth are carried inwards with the bullet and scattered in the superficial part of the wound track, whereas the heavier missile becomes buried deeply in the cerebral tissue, and thus two widely separated foci of possible infection are introduced, either of which may lead to abscess or spreading encephalitis (Fig 32)

CHAPTER II

PATHOLOGY

INTEREST in disease of the brain started, as far as is known, in the neolithic age many thousands of years ago, when holes were rubbed into the skull by flints to let out those demons which to-day would be designated by more formal pathological terms

Actual reference to injuries of the skull was made as early as 1600 B.C. in a papyrus [1] written by a surgeon who knew that the



FIG 33
John Hughlings Jackson

pulse may be affected by a head injury, that the patient may sustain a depressed fracture of the vault even though the scalp was not torn, and that such injury, if it involved the brain, might cause loss of speech and paralysis of the limbs. Hippocrates [2] chronicled his experiences on wounds of the head in the fifth

[1] Garrison, F H. "History of Medicine," 155 Philadelphia, 1929

[2] Hippocrates "Wounds of the Head" Translation in Loeb Classical Library, 1927, 3. Edited by Capps, Page and Rouse

century B C and in his discussion on operative treatment warned the surgeon not to injure the brain otherwise convulsions and palsy would ensue This advice no doubt was found useful later by Galen, for whom Roman arenas provided a great number of cases on which to practise the art of trepanning According to Garrison,[1] "Lanfranchi of Milan was the first to describe concussion of the brain, and his chapter on symptoms of fracture of the skull is accounted a classic." In A D 1170, Roger of Palermo[1] published his "Practica Chirurgica," of which the first part is



FIG 34
Sir Victor Horsley

concerned with wounds of the head and fractures of the skull He advised that a finger should be introduced into open wounds and moved over the underlying bone so as to discover the presence of a fracture, and this practice is still in use to-day Since those early days a voluminous literature on cerebral and cranial injuries has accumulated and advanced in scientific importance as more accurate methods of observation were discovered, such as microscopy by Jansen in 1590 The correlating of clinical signs with post mortem findings became firmly established at the end of the eighteenth century, and this practice combined with newer

[1] Garrison, F H. "History of Medicine 152, 155 Philadelphia, 1929

experimental methods, led to great additions to knowledge by a succession of brilliant neurologists in the following century.

John Hughlings Jackson, the son of a Yorkshire farmer, served his apprenticeship at the York Medical School and St Bartholomew's Hospital before he qualified in medicine in 1856 at the age of twenty. "His gift was a subtle one. It guided thought into new channels, and even to-day the penetrating surmise of this clinical philosopher continues to provide the modern student of the nervous system with constructive hypotheses." [1]



Fig 35
Dr Harvey Cushing

At the Queen's Square Hospital he made those detailed and accurate clinical observations correlated with post-mortem findings from which he adduced the localisation of function in the cerebral cortex and, more important, the general integration of function of the whole nervous system. The names of Fritsch and Hitzig of Germany and Ferrier of London should be mentioned in association with Jackson in the localisation of cortical function.

In 1878 Victor Horsley passed his examination for fellowship of the Royal College of Surgeons. It was chiefly through his long and brilliant work that the surgery of the nervous system was

[1] "Neurological Biographies and Addresses" Oxford University Press 1936

set on solid and scientific foundations Amongst other things, he discovered how to open the skull safely and that bleeding from bone could be controlled by rubbing beeswax into the diploic openings It is of interest to note that, though not directly concerned with cerebral trauma, he was the first to operate on and to remove successfully a spinal tumour This gives some idea of the advance of neurosurgical methods even in the year 1887 [1]

Harvey Cushing graduated in medicine at Yale University, U S.A., although it was as Professor of Surgery at Harvard that he conducted his brilliant neurosurgical clinic at the Peter Bent Brigham Hospital in Boston Cushing undoubtedly is the father of modern neurosurgery To him and indirectly to his mentor Halsted we owe the meticulous and life-saving technique of present-day neurosurgery He died in 1939, but the Cushing traditions will never die [1]

The advances in neurology and neurosurgery certainly throw new light on cerebral trauma, but none the less this problem has by no means been solved in spite of the interest it has aroused, and to-day circumstances are making the subject the concern of everyone engaged in traumatic surgery or medicine, not only because of the abundance of material but also because of its widespread distribution

The following table indicates how many people are killed each year on the British roads, and of these a large percentage die as the result of head injuries Furthermore, for every fatality due to cerebral trauma at least four other people receive non fatal injuries to the skull or brain which lead to prolonged morbidity

ROAD ACCIDENTS

Year	Killed	Injured	Total
1027	5,320	148 575	153 904
1036	6 561	227 813	234,373
1037	6 633	226 402	233 035
1038	6 648	226 711	233,359
1039	8,272		
1040	8 609		
1041	9 169	146,327	155 696
1042	6 926	140 618	147,544
1043	5 796	116 740	122,536
1044	6 416	124 458	130 874
1045	5,256	133 042	138,298
1046	5 062	157 484	162,546
1047	4,881	161,318	166 199

Ministry of Transport figures.

[1] "Neurological Biographies and Addresses. Oxford University Press 1936.

These are important facts, and in order to give further perspective to the subject the relevant details of a clinical series of 1,000 cases and the pathological findings of fifty consecutive post-mortem examinations will be found in tabulation form below.

A THOUSAND CONSECUTIVE CASES OF ACUTE INJURY OF THE HEAD

Age in Decades.	Sex		Fractures with the Scalp Intact.			Compound Fracture of the Vault.		Associated Injuries.				Type of Accident.			Number of Deaths.	
	M	F	X ray Evidence of Fracture	Clinical Evidence of Fracture.	No Evidence of Fracture	Linear	Depressed.	Spine	Limbs	Thorax.	Abdomen.	Road.	Industrial.	Other Types.	M	F
1	84	48	57	35	42	3	6	0	16	6	0	72	0	60	7	4
2	160	36	82	32	74	5	7	0	18	8	0	144	38	14	19	7
3	172	32	75	50	73	6	8	0	12	6	0	136	28	40	24	5
4	112	44	75	30	51	8	5	2	10	4	2	84	52	20	12	9
5	80	28	66	22	16	9	3	2	14	2	2	68	28	12	15	6
6	48	48	52	21	25	3	2	0	4	0	1	48	24	24	11	11
7	44	20	37	17	17	1	0	2	12	2	0	40	8	16	9	7
8	24	20	28	7	16	2	0	2	6	4	0	20	0	24	8	9
TOTAL	724	276	472	214	314	37	31	8	92	32	5	612	178	210	105	58
Percentages	72·4	27 6	47 2	21 4	31·4	3 7	3 1	0 8	9 2	3 2	0 5	61 2	17 8	21·0	10 5	5 8

NOTE —This series does not include war or air raid casualties.

NUMBER AND TYPE OF OPERATIONS PERFORMED IN THE ABOVE SERIES

Type of Operation	Recovered	Deaths
For extradural hæmorrhages	9	2
For acute subdural hæmorrhages	2	3
For chronic subdural hæmorrhages and hygroma .	3	0
Decompressions for raised intracranial tension	5	2
Exploratory trephine openings	10	3
For compound depressed fractures	14	7
Excisions of compound linear fractures	5	2
To raise depressions in closed fractures	3	0
Total number of operations	51	19

NOTE —This list does not include repairs of simple wounds of the scalp which were multitudinous Spinal drainages or other methods of dehydration are also omitted, as these were routine measures in many of the cases

Two meningeal hæmorrhages not operated on were found at autopsy .—

(*a*) One was diagnosed, but the patient died before operation could be performed

(*b*) One was not diagnosed because there was no latent interval. This was complicated by a profuse subarachnoid hæmorrhage

CAUSES OF DEATH IN ABOVE SERIES

Cause of Death.	Within Twelve Hours	Within Twenty-four Hours.	Second Day	Third Day	Fourth Day	Fifth Day	Sixth Day	Seventh Day	Second Week	Third Week	Later	Total No. of Deaths due to each Cause.	Percentage of Deaths due to each Cause.
Contusion	47	54	15	0	1	2	1	2	2	1	0	134	82·0
Meningitis	0	0	1	1	3	0	0	0	3	1	1	9	5·5
Pneumonia	0	0	2	2	3	1	0	0	1	0	2	11	6·7
Associated Injuries	3	0	3	1	0	0	0	0	0	0	0	6	3·6
Exhaustion	0	0	0	0	0	0	0	0	0	2	0	2	1·0
Loss of blood	0	0	0	0	1	0	0	0	0	0	0	1	0·6
Total number of death in 1,000 cases	50	54	21	12	7	3	1	2	6	4	3	163	
Percentage of deaths in 1,000 cases	5·0	5·4	2·1	1·2	0·7	0·3	0·1	0·2	0·6	0·4	0·3		

Percentage fatality rate, 16·3.

FIFTY CONSECUTIVE AUTOPSIES

	Brain Tissue Undamaged Macroscopically	Lacerations.		Surface Contusions		Intracerebral Hæmorrhages		Subarachnoid Hæmorrhages	
		Superficial	Deep	Single	Multiple	Petechial	Massive	Slight	Profuse
Number of cases in which each lesion occurred	2	9	5	18	24	23	1	9	27
Percentage occurrence	4	28		84		48		72	

	Acute Subdural Hæmorrhages		Extradural Hæmorrhages		Meningitis	Fractures of the Skull				Associated Injuries.
	Slight	Profuse	Thin Layer	Large Clot.		Vault	Base	Both.	Neither.	Severe.
Number of cases in which each lesion occurred	3	4	7	3	3	5	4	36	5	15
Percentage occurrence	14		20		6	10	8	72	10	30

The autopsy group was collected recently from several sources and includes material partly from clinics not under my direction and partly from the later fatalities of the above clinical series.

In the two cases in which no damage occurred to the brain tissue extensive surface hæmorrhages were found—one extradural and the other subdural Surface contusions occurred most commonly on the under and lower parts of the outer surfaces of the hemispheres and frequently in the cerebellum Severe lacerations were usually found under displaced fractures, whether these were open or closed In one case the hypothalamus had been severed transversely The most profuse surface hæmorrhages were due either to tearing of the parasagittal sinus or to the veins which drain into it.

There were ten cases of extradural hæmorrhage Of these seven were thin clots and, judging by the severity of lesions

elsewhere in the body, were of little significance. In three cases a massive clot was found: one, due to basal rupture of the meningeal vessels, was uncomplicated by lesions elsewhere in the brain; the others—one due to tearing of the sagittal sinus and the other to diploic bleeding—were complicated by other cerebral lesions. Fracture of the skull occurred in 90 per cent. of cases and in 72 per cent. both the base and vault were involved. When the skull was not fractured, contusional lesions of the brain were not as pronounced as in fracture cases, but surface hæmorrhages were equally profuse. Severe injuries elsewhere in the body were common. Subgaleal bleeding was also common.

THE PATHOLOGY OF CLOSED INJURIES OF THE BRAIN

The Three Primary Pathological States

During the few moments the accidental forces are operating the brain can be damaged in three and in only three ways. It



Fig 36

A small contusion on the under surface of the right cerebellar hemisphere caused by movement of the brain within the skull

may be contused or lacerated or its neurones may undergo a diffuse neuronal injury of submicroscopical dimensions. One or any combination of these conditions may occur. All other phenomena such as œdema and massive hæmorrhages are secondary, even though they may develop immediately afterwards.

Contusions—Cerebral contusions are microscopical solutions of continuity of brain tissue. They may occur on the surface of the brain (Fig 37) or deep within its tissue, and both types are necessarily associated with bleeding, though this is usually punctate and limited to the area of the bruise. When superficial they are seen as subpial gelatinous looking stains of a mottled reddish blue colour covered by a thin layer of blood (Fig 36). Thick clots cannot occur because the pia is firmly attached to the brain

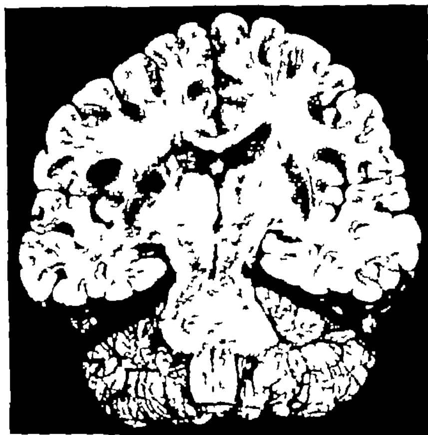


Fig. 37
Petechial hæmorrhages.

tissue and will not readily strip. Often contusions are wedge-shaped, with the apex of the wedge extending into the cerebral tissue for a distance of 1 or 2 cm, but rarely deeper than this.[1] They may be single or multiple, any surface or more than one surface of the brain may be involved including the upper aspect of the corpus callosum, and they may be no bigger than a pinhead or may cover large areas on both sides of the brain.

Within the brain substance, contusions are seen as clusters of petechial hæmorrhages superimposed on a dull grey background

[1] Courville, C. B. "Pathology of the Nervous System," Part IV 223. Pacific Press, California, 1937.

(Figs. 37 and 38). They occur in the hemispheres, both in the white and grey matter. They are also found in the cerebellum (Fig. 39), medulla, pons,[1] midbrain, thalamus, hypothalamus, caudate nucleus, lenticular nucleus and in the subependymal layers of the ventricles and of the aqueduct of Sylvius They may be single or widespread in distribution.

Microscopically, either superficial or deeply placed contusions show as spots or streaks of extravasated blood surrounded by an area of damaged brain cells, beyond which is a limited zone of œdema [2] The extravasated blood is due either to ruptured vessels

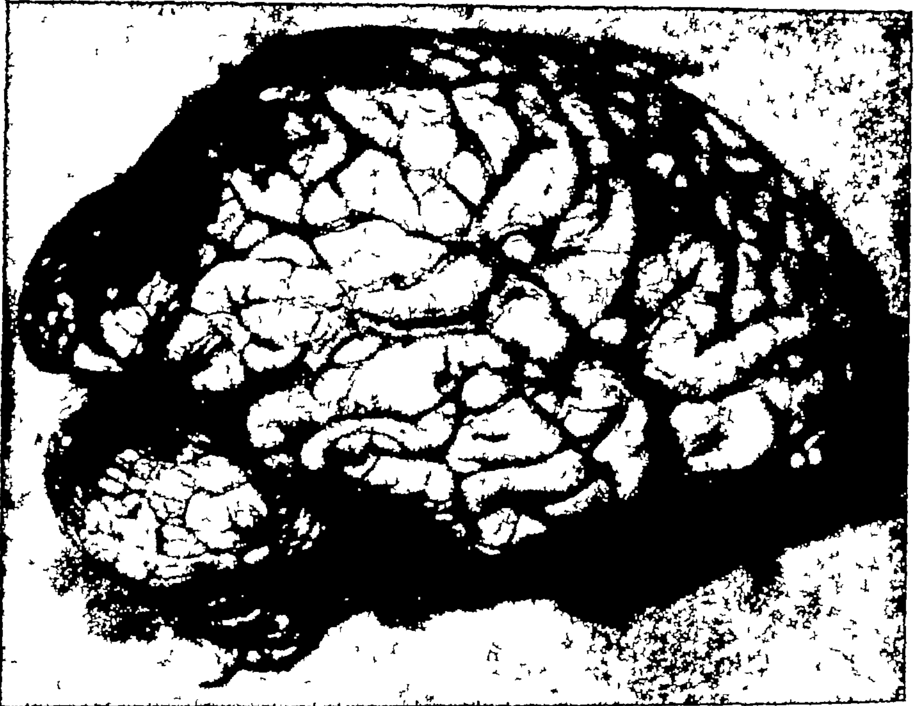


Fig 38
Occipital bruising

or diapedesis. The area of damaged brain cells is composed of three concentric zones, each one being distinguishable from the other by the nature of the cellular changes within them. Nearest to the extravasated blood the neurones, glia and microglia are completely destroyed and often are in a state of liquefaction. In the middle zone the staining properties and shapes of the cells are altered in such a way that it is impossible to know whether the cells have been permanently injured or not. At the periphery

1 Greenfield, J G "Some Observations on Cerebral Injuries" *Proc Roy Soc Med*, 1938, 32, 43-52

2 Rand, C W, and Courville, C B "Histologic Studies of the Brain in Cases of Fatal Injury to the Head (6) Cyto-architectonic Alterations" *Arch Neur and Psych.*, 1936, 36, 1277-1293



Fig 39
Petechial hæmorrhages within the brain stem and cerebellum.

the neuroglial astrocytes and the microglial cells are hypertrophied or increased in numbers, and it is from this zone of increased cellular activity that the processes of phagocytosis and repair are initiated



Fig 40
A laceration of the outer surface of the right temporal lobe associated with a subarachnoid hæmorrhage

Many of the cells within a contusion, though functionally paralysed, are viable and will recover under favourable conditions or succumb if the cerebral circulation is impaired by secondary development such as œdema

Laceration.—Lacerations are gross solutions of continuity of brain tissue and differ from contusions merely in their severity



FIG 41

A frontal injury with laceration of the frontal lobe

Most commonly they are found on the under surfaces of the frontal lobes and near the tips of the temporal poles (Fig 40). Usually they are superficial, but may be deep enough to open into a ventricle or cut across a basal nucleus such as the hypothalamus. Deep lacerations are invariably associated with displaced fractures and may occur anywhere. Contusions of the choroid plexuses and walls of the ventricles are common. Lacerations of these structures, on the other hand, are rare.

The pia is invariably torn in a laceration, which means that ruptured cortical vessels may bleed with little restriction into the voluminous subarachnoid spaces Tearing of the arachnoid membranes also is usual and results in blood and cerebrospinal fluid leaking into the subdural space The tough dura, however,



Fig. 42
Severe laceration of the temporal lobe

rarely gives way and thus minimises the incidence of post traumatic epilepsy as will be seen later

Shaggy walled cavities (Figs. 41 and 42) distended with blood are found in severe injuries, and occasionally whole lobes of the brain may be so thoroughly disintegrated that the tissue may be washed away with jets of water In severe crushing injuries complete pulping of the brain may occur

The Healing of Contusions and Lacerations.—The processes of repair in the brain are similar in principle to those elsewhere in the body, inasmuch as dead tissue has to be removed and the wound consolidated. The histopathological details, however, are peculiar to the specialised cerebral tissues, and it is of interest to know what the possible final results of a wound may be.

There are three types of cell in the brain (Fig. 43).—

(i) The neurones }
(ii) The neuroglia }—of ectodermal origin.
(iii) The microglia—of mesodermal origin

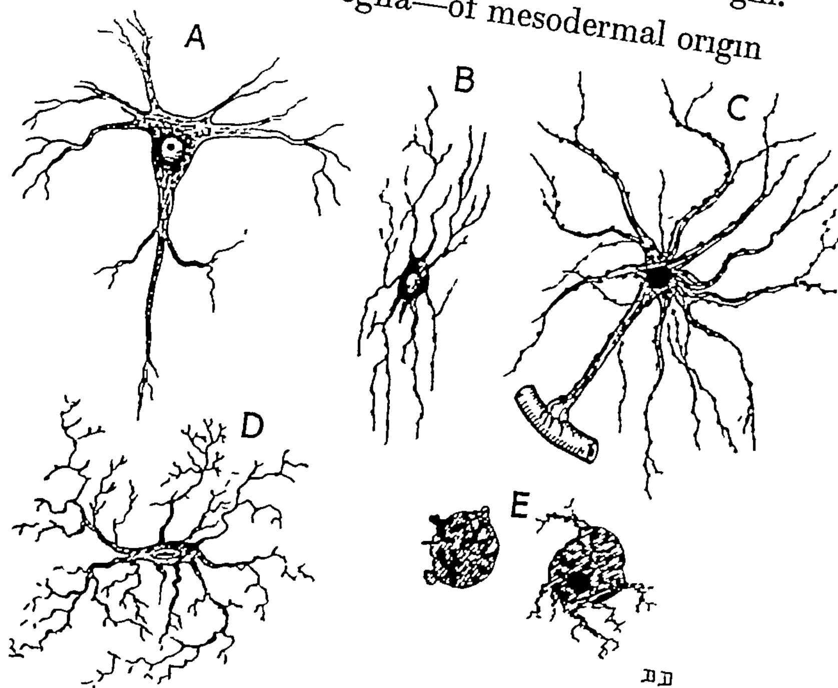


FIG 43

The types of brain cell A, neurone, B, oligodendrocyte, C, astrocyte, showing sucker foot, D, microglial cell in resting phase, E, microglial cell in active phase (Gitterzellen)

The neurones are the cells which initiate and transmit the nerve impulses. When they die it is believed that they are never replaced either from embryonal sources or by division of a neighbouring cell, although it is possible for a neurone to recover its function after a period of prolonged inactivity.

The neuroglia is an interstitial tissue, but none the less a fundamental part of the nervous system, and together with the blood vessels supports the brain and gives it cohesion Probably it is essential to neuronal metabolism. It is composed of two types of cells, the astrocytes and oligodendrocytes. The astrocytes are stellate in shape and have dendritic processes issuing from their angles, one of which, known as the sucker foot, is longer and

thicker than the others, and is attached to the wall of a blood vessel The oligodendrocytes are round or oval cells containing large nuclei which almost completely fill their protoplasm, and their processes are fine and branched From their arrangement along the axons of the nerve cells it is presumed that they are principally concerned with the metabolism of myelin

The microglia was so named by Hortega,[1] who proved that certain interstitial cells were neither astrocytes nor oligodendrocytes

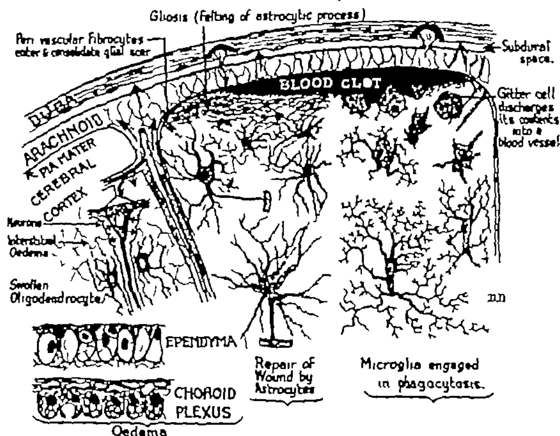


FIG 44

Repair of a cerebral wound. On the left side of the picture is shown an area of oedema The perineuronal and perivascular spaces are distended with fluid and the cells of the ependyma and choroid plexus are enlarged and vacuolated. The uncharted arrows indicate the circulation of the tissue fluids.

and that they possess migratory and phagocytic powers In the resting phase these cells have a dark nucleus surrounded by scanty protoplasm, and possess two or more slender processes which are feathery in appearance owing to short side branches Presumably they are part of the reticulo-endothelial system and play an important rôle in all inflammatory and necrotic processes of nerve tissue

The first process in repair is liquefaction of dead tissue and extravasated blood cells and the various stages of this activity

[1] Rio-Hortega, P Del "The Microglia. *Lancet* 1939 1, 1023.

may be seen in a contusion as it changes in colour from dark red through lighter shades of brown to a yellowish tinge. Physico-chemical phenomena account for liquefaction, and it has been suggested that the chemical agents concerned may come either from the damaged cells themselves or from the blood stream.

At an early stage microglial cells from the surrounding brain tissue invade the damaged area and engulf the lipoids produced by the liquefaction, finally depositing them in neighbouring blood vessels or in the subarachnoid spaces. When actively engaged in phagocytosis the cells change their shape; their processes are retracted and their bodies are swollen and granular. When in this condition they are known as scavenger cells, compound granular corpuscles or "Gitterzellen," and are most numerous on the sixth day following an injury.

According to Linell,[1] hypertrophy of the astrocytes surrounding a contusion is evident at the end of three days, and in three weeks these spider cells have migrated in considerable numbers into the wound. This condition is known as gliosis. In four weeks, felting of the astrocytic processes has occurred, and in two months cell bodies have disappeared and a firm glial scar remains, the extent of which depends on the amount of dead tissue which had to be removed. A clean-cut wound, for example, as Penfield[2,3] has shown, will heal with less gliosis than a lacerated wound with ragged edges. In simple contusions, where there has been no gross loss of cerebral tissue, the final healing may be obtained purely by glial processes. In lacerations, or in contusions where there has been gross loss of tissue, the final cementing of the wound is brought about by fibrous tissue which grows in from the mesodermal elements of neighbouring blood vessels and particularly from those on the surface of the brain (Fig. 44) The manner in which fibrous tissue grows in from the surface explains why so many scars are attached to the leptomeninges. In those cases where the loss of cerebral tissue has been great, cysts lined with fibrous tissue and containing xanthochromic fluid may be found within the gliocicatrix, and these no doubt are nature's economical way of filling in space.

A scar may or may not be the final result of a contusion or laceration. Occasionally a degeneration of the nervous elements takes place after an interval of a few weeks or even years, which proceeds far beyond the limits of the original contusion and leads

[1] Linell, E A "Histology of Neurological Changes following Cerebral Trauma Experimental Investigation" *Arch Neur and Psych*, 1929, **22**, 926-948

[2] Penfield, W "The Mechanism of Cicatricial Contraction in the Brain" *Brain*, 1927, **50**, 499

[3] Penfield, W, and Buckley, R "Punctures of the Brain The Factors concerned in Gliosis and in Cicatricial Contraction" *Arch Neur and Psych*, 1928, **20**, 1

to all kinds of neurological disorders Also progressive softening may result in delayed intracerebral hæmorrhage This happening was described by Bollinger[1] as late traumatic apoplexy, and is



FIG 43
Severe cortical atrophy following trauma.

of particular importance when considering the possible relationship of a sudden cerebral crisis to previous cerebral trauma.

The condition of "punch drunk" is well known in professional boxers and is due to degenerative changes in the brain consequent

[1] Bollinger O *Internationale Beiträge zur Wissenschaftlichen Med. Festschr* Rudolf Virchow's Vollendung seines 70 Lebensjahres, 2. Berlin, 1891

on small repeated hæmorrhages or contusions which, in themselves, are of no particular importance [1,2]

In syphilitics or arteriosclerotics a degenerative encephalopathy



FIG 46

This is the type of closed fracture in which the meninges are torn and cerebrospinal fluid escapes into the subgaleal space to produce a traumatic meningocele It is also the type of fracture which acts as a natural internal decompression, as it allows blood to escape from the extradural into the subgaleal tissues, thereby avoiding compression of the brain It is the type of fracture most frequently seen in children, and explains how extensive calvarial fractures often occur without signs of severe cerebral compression

may be precipitated by an injury, or a pre-existing diseased state may be aggravated and its pathological processes accelerated.

[1] Martland, H S "Punch Drunk" *J A M A*, 1928, **91**, 1103

[2] Parker, H S "Traumatic Encephalopathy (Punch Drunk) of Professional Pugilists" *Jour Neur and Psych*, 1934, **15**, 20

Cortical atrophy is often the final result of a severe injury to the brain as proved by encephalography done in the routine investigation of post concussional symptoms, when the air studies show that the loss of tissue is compensated for by dilatation of the lateral ventricle which appears to wander into the hemisphere concerned (Fig 45)

On those rare occasions when a closed linear fracture is associated with a tear in the underlying dural and arachnoid membranes, cerebrospinal fluid escapes into the subgaleal space, and a cyst communicating with the subarachnoid cavity may develop beneath the scalp (Figs 46 and 47) Such a condition



FIG. 47

The beginning of a traumatic meningocele The swelling on the right side of the head is due to an escape of bloodstained cerebrospinal fluid beneath the scalp.

is known as a traumatic meningocele and occurs more commonly in children, possibly because the firmer attachment of the dura to the bone in the young renders it more liable to tearing than its looser attachment in adults The wall of the meningocele is non absorbable and results from reaction of fibrous tissue cells lining the scalp to the irritation of the cerebrospinal fluid Such reaction to the presence of cerebrospinal fluid always occurs in non neural tissues and is a manifestation of the inherent tendency of the nervous system to insulate itself This tendency to neural insulation is of great surgical importance, as it explains the failures of operations designed to relieve hydrocephalus by drainage of the cerebrospinal fluid into the subcutaneous or submuscular spaces When an opening through the dura and skull is wide,

or when large areas of bone disappear owing to malacia (Fig. 48), brain tissue may herniate into the meningocele and convert it into a meningo-encephalocele or into a meningo-encephalo-ventriculocele if the ventricle is also included in the brain hernia.

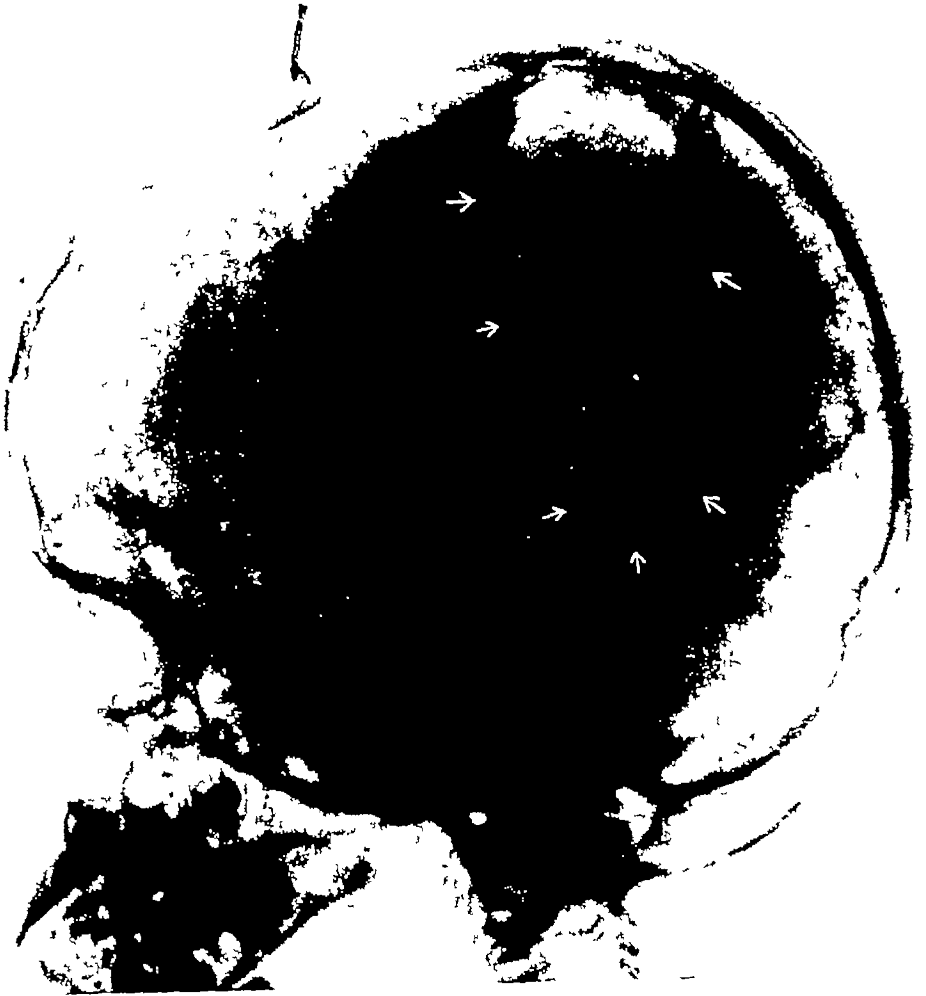


FIG 48

A case of traumatic malacia or absorption of bone following trauma The scalp was not broken at the time of injury

DIFFUSE INJURIES

Contusions, lacerations and hæmorrhages commonly found at autopsy, or on surgical exploration after acute head injuries, often will not account for unconsciousness with its allied neurological states. Therefore, cells or pathways concerned in the initiation or transmission of nervous impulses other than those in the areas which have undergone damage of macroscopical

dimensions must have been affected by the injury The actual nature of the changes in the neurones concerned constitutes one of the main problems of acute cerebral trauma and, in view of the great controversy the subject is arousing, it is better to avoid the use of terms such as commotio cerebri, which imply that the physiopathology of diffuse traumatic neural paralysis not due to gross contusion or compression is known The term "concussion," although sometimes used to mean that the brain tissue itself has been concussed—a hypothetical form of injury—is best restricted to a clinical sense denoting that the state of consciousness has been impaired as the result of a mechanical force applied to the head Much experimental work has been carried out in an attempt to elucidate the neuropathology and neurophysiology of concussion, and to-day there are three main theories

The Vascular Theory.—Trotter,[1] basing his ideas on the work of Strohmeyer[2] and of Kocher,[3] believed that in acute distortions of the skull the capillaries of the brain are obliterated by compression, and he suggested that the resulting cerebral anæmia accounted for unconsciousness. The sequence of neurological events in non fatal cases he ascribed to the gradual recovery of the circulation, which goes through a phase of venous congestion leading to hyperexcitability of the neurones The simplicity of this theory has made it attractive and it has been widely accepted, although explanations of the cerebral anæmia other than by compression, such as widespread tearing of the neuromuscular apparatus of the arterioles or excessive stimulation or paralysis of the vasomotor centre, have been offered

According to Ricker,[4] damage to the neuromuscular mechanisms of the cerebral blood vessels first of all causes excessive vascular dilatation with consequent slowing of the blood stream and then extreme constriction which leads to ischæmia. In fatal cases of cerebral trauma widespread vascular changes in the brain may often be demonstrated histologically, and Winkelmann and Eckel[5] have shown that the capillaries as well as the venules are congested, which suggests that the vascular changes are the result of active participation of the vessel walls in this state rather than the consequence of pure mechanical obstruction due to increased intracranial pressure. Swelling of the endothelial cells is an early occurrence, and from this stage up to complete occlusion

[1] Trotter W "Injuries of the Skull and Brain": Choyce "System of Surgery" 8, 338. Cassell & Co. London, 1932. "An Address on the Evolution of the Surgery of Head Injuries." *Lancet* 1930, 1, 169. "An Address on the Management of Head Injuries." *Lancet* 1933 2, 933.

[2] Strohmeyer L. *Handbuch der Chirurgie* Bd. 11 Freiburg

[3] Kocher T *Nothnagel's specielle Pathologie u. Therapie.* IX Wien 1901

[4] Ricker G *Virchow's Arch. f Path. Anat u. Physiol u. für Klin. Med.*, 1919 226, 180.

[5] Winkelmann, N W., and Eckel, J L. "Brain Trauma. Histopathology during the Early Stages." *Arch Neur and Psych.*, 1934 31, 956

of the lumen of the vessel all degrees of endarteritic change have been described.

It will be seen, therefore, that though microscopical evidence cannot prove that cerebral anæmia is responsible for the production of the phenomena of concussion, it does show that diffuse vascular changes *are* present in the brain, and these presumably are important. Certainly it is stasis of the blood-flow which accounts for small foci of softening and for those areas from which the neurones so strangely disappear.[1]

Theory of Physiological Neuronal Injuries.—For many hundreds of years it has been known that concussion may be caused by a slight shake up of the head which does not produce a demonstrable cerebral injury and is unassociated with signs of violence, either in the skull or in the overlying integuments.

In 1705 Littre [2] published a case which stimulated widespread interest in the pathology of concussion, and it is from this time that the conception of this state took on some kind of definite form. Littre's case was that of a strong young criminal who, wishing to forestall his sentence of the rack, took his measure of the cell where he was confined and, crouching with hands behind his back, ran towards the opposite wall, hitting his head against it with all his strength. He fell dead on the spot without saying a word or uttering a single cry

M. Littre, called to inspect the corpse, began by examining the outside of the head. He was surprised to find there was no contusion, open wound or fracture. He then cut and separated all the integuments of the skull at the top of the head where, according to the evidence of several other criminals who had witnessed the act, the impact had occurred. He examined these integuments also from inside and found there no more alteration than had been found on the outside. He did not notice any injury to the skull bones after having uncovered them, except that the scaly part of the right temporal bone was separated from the parietal for a short distance.

It was therefore necessary for him to saw the skull and examine the brain. This he did, but M. Littre's surprise grew when he found everything in a quite normal state and perfectly healthy. The only unusual thing was that the brain did not nearly fill the whole interior capacity of the skull, and its substance, as well as that of the cerebellum and spinal cord, was firmer and more solid to the sight and touch than normal. M. Littre made more certain of the obvious cerebral shrinking by putting the cut pieces

[1] Howkins, J, M'Laughlin, C R, and Daniel, P "Neuronal Damage from Temporary Cardiac Arrest" *Lancet*, 1946, April 6, p 488

[2] Littre "Diverses Observations Anatomiques" *Hist de l'Acad Roy des Sciences*, 1705, 54.

of brain and the skull back in their place, which he was able to do very easily, although in other cases this could only be done with great difficulty

His conclusion, therefore, was that the brain had sunk in very considerably with the violent disturbance of the blow, and as it had little elasticity it was not able to spring back to its former position Consequently the distribution of nervous impulses in the remainder of the body, essential for all movements, had ceased at once

As the report of this case is so important it is thought advisable to reproduce it in the above manner rather than to paraphrase it.

In 1866 Erichsen[1] came to the conclusion that spinal concussion was due to a molecular disarrangement of the cells concerned, and this probably led to the theory of cerebral concussion known as commotio cerebri or commotion of the brain cells

To-day it is a firmly accepted belief that the brain can receive a purely non structural physiological injury sufficient to produce a diffuse neuronal paralysis This belief, of course, is of paramount importance, as it must influence profoundly any attitude in regard to treatment of concussion, since anything mechanistic such as decompression is unlikely to improve this state. The evidence in support of the physiological theory is largely forthcoming from experiments in which the brains of animals whose heads have been subjected to small repeated blows sufficient to cause concussion have shown negative macroscopical or microscopical findings[2]

Recently Brown and Russell[3] have proved by experiment that concussion may be caused by the forces of acceleration alone, that the resulting neurological state is of the non structural type and that unconsciousness is probably due principally to injury of the medulla.

The Theory of Organic Neuronal Injury.—The essential principle of this theory is that unconsciousness and allied neurological states can be explained by demonstrable changes in the neurones themselves, which means that concussion is based on morbid anatomy[4] From the nature of any severe accident in man it is reasonable to assume that diffuse and organic neuronal changes must be frequent, since the head is often subjected to severe violence sufficient to deform the whole brain, and deformity implies bending, stretching or tearing Histologically, neuronal

[1] Erichsen, J E. "On Concussion of the Spine, Nervous Shock and other Obscure Injuries to the Nervous System." New Edition. Wm. Wood & Co. Baltimore, 1886.
[2] Jakob, A. *Nissl Alzheimer's Histologie u. histopath Arbeiten über die Grosshirnrinde.* Jena 1912, 5, 182.
[3] Denny Brown, D E., and Russell, W R. "Experimental Concussion." *Proc Roy Soc Med.*, 1941 34, 691
[4] Tedeschi, C G "Cerebral Injury by Blunt Mechanical Trauma." *Arch Neurol* and *Psych.*, 1945, 53, 133.

changes distant from the sites of contusion have been proved by the studies of Rand and Courville,[1] and Greenfield,[2] though the latter writer believes that they might be secondary to œdema. In my opinion, demonstrable changes in neurones other than in contusional areas can always be found following fatal injuries, and particularly if the patient has survived for more than twenty-four hours. These findings suggest that concussion may be due to organic neuronal injury, but nothing more definite can be claimed for them since correlation of loss of function with anatomical change is always conjectural. Furthermore, the distribution and exact number of injured neurones necessary for the production of the phenomena of concussion are not known, and therefore at this stage it would be opportune to consider which part of the brain is concerned with consciousness and what is meant by unconsciousness. Although unconsciousness is often appreciable at a glance its definition is extremely difficult, and possibly new words will have to be coined before a completely satisfactory one is produced. In unconsciousness there is a cessation of those mental processes which normally can be engaged by an observer. Whether the patient is totally unable to think even in semicoma is not known, but judging from restlessness some form of intellectual activity must remain, otherwise his movements can only be regarded as a form of epilepsy of the postural centres. Moreover, a semiconscious patient, driven by the discomfort of a distended bladder, may attempt to get out of bed to relieve himself, and this is indicative of a complex mental process rather than of a pure reflex phenomenon. Reactions to external environment are changed, and in particular an unconscious patient is unresponsive to stimulation of sight and hearing. Muscle power and co-ordination may be largely retained, but the patient seems unwilling to use his limbs except for purposes of defence These various points are embraced in Mapother's [3] definition of consciousness, which is an awareness of external environment and accessibility. According to Russell,[4] full consciousness means that any occurrence in which the patient is actively or passively concerned makes an impression on his memory which can subsequently be brought to mind, and thus the length of any state of unconsciousness can be measured by the length of post-

[1] Rand, C W, and Courville, C B "Histologic Studies of the Brain in Cases of Fatal Injury to the Head (5) Changes in the Nerve Fibres" *Arch Neur and Psych*, 1934, **31**, 527-555

[2] Greenfield, J G "Some Observations on Cerebral Injuries" *Proc Roy Soc Med*, 1938, **32**, 43-52

[3] Mapother, E "Mental Symptoms associated with Head Injury The Psychiatric Aspect" *Brit Med Jour*, 1937, **2**, 1055-1061

[4] Russell, W R "Cerebral Involvement of Head Injury" *Brain*, 1932*a*, **55**, 549. "Discussion of the Diagnosis and Treatment of Acute Head Injuries" *Proc Roy Soc. Med*, 1932*b*, **25**, 751

traumatic amnesia. Sleep and unconsciousness are very similar clinical states, though they arise from very different causes. Sleep is the result of the integration of numerous physiological processes, whereas the basis of traumatic unconsciousness is morbid and possibly anatomical.[1] It has not been finally decided whether there is a centre for consciousness, but, judging by the work of Penfield,[2] lesions about the thalamus are more apt to lead to unconsciousness than lesions elsewhere in the body. Furthermore, Dott[3] has shown that tumours in the anterior end

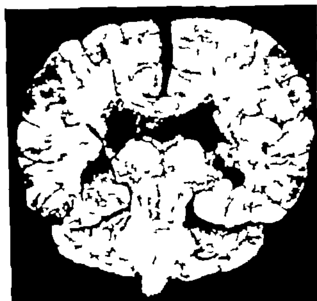


FIG. 40

In this case the midbrain had been virtually rocked from the cerebral hemispheres (see Greenfield's "Theory of Concussion.")

of the third ventricle when involving the hypothalamic nuclei are occasionally associated with unconsciousness. Duret[4] was of the opinion that concussion is due to injury of the walls of the third ventricle and of the aqueduct of Sylvius and of the fourth ventricle by a wave of cerebrospinal fluid travelling forcibly from its site of origin in the lateral ventricles towards the spinal canal.

According to Greenfield, sudden displacements of the hemispheres in relation to the incisura tentorii deform or stretch the connections of the hemispheres with the brain stem, resulting *either in damage to the thalamus or to isolation of the hemispheres.*

[1] Kleitman, N. "Sleep and Wakefulness." Chicago, 1939.
[2] Penfield, W. "Cerebral Cortex in Man: Cerebral Cortex and Consciousness (Harvey Lecture)." *Arch. Neur. and Psych.*, 1938, 40, 417-442.
[3] Dott, N. M., Le Gros, Clark, and Riddoch, G. "Hypothalamus." Oliver & Boyd, Edinburgh, 1938.
[4] Duret, H. "Traumatismes cranio-cerebraux." Paris, 1920.

This theory of "deformation and stretching" is an extremely attractive one and explains the underlying cause of many cases of concussion. It does not, however, decide finally whether consciousness is centred in or on the thalamus.

Jefferson[1] regards traumatic stupor or unconsciousness as a form of unnatural sleep. Therefore he suggests that the word "parasomnia" be substituted for these terms as it more nearly connotes the true underlying neurophysiological processes which bring about the clinical state.

He believes that concussion is neurogenic and not vascular in origin, but is driven to the conclusion "that there is no single factor or group of facts which will tell us the cause of unconsciousness in head injuries." From other sources, and particularly from cases of non-traumatic local bleeding, he deduces that traumatic stupor is due essentially to injury to the brain stem or hypothalamic areas. He does, however, state that it is his belief that *important cortical disturbances do occur in concussion* and affect the final clinical picture. No one, of course, doubts that brain stem injury can cause coma (Fig. 50)

It is obvious that the phenomena of concussion must be due to alterations in the neurophysiological activities of the neurones, either in the nature of paralysis or of hyperactivity, and probably both states exist at different neurological levels. Moreover, the injury may strike hardest at the cells themselves where the nervous impulses are initiated, or at their axons along which the impulses are conducted, and thus widely separated lesions can give rise to the same neurological signs.

In my opinion concussion in human beings is due in the majority of cases to paralysis or dysfunction of the cortical cells or to blockage of the subcortical pathways and, more rarely, to local injury within the basal ganglia or brain stem. With regard to stupor, my feeling is that, amidst all the evidence of neural paralysis, the one structure that remained alive was the brain stem and basal nuclei. A patient cannot hear, speak, see or think normally, yet all those complex functions which are relegated by traditional neurology to the brain stem and basal nuclei are functioning normally. The heart, in most cases, is regular, although perhaps the rate may be a little fast; respiration is usually regular and usually normal in rate. In the large majority of cases metabolism of sugar, fat, heat regulation, etc., is being carried on efficiently and often normally.

In concussion a patient's eyes may be open and as far as we can judge he does not see, in a way that suggests that the cortical visual pathways are paralysed. On the other hand, the light reflex is

[1] Jefferson, G "The Nature of Concussion" *Brit Med Jour*, 1944, 1, 1

briskly active, which shows at least that the reflexes concerned with the basal nuclei or upper part of the brain stem are alive The mental changes which are met with in the recovery stages of confusion surely could not be evidences of brain stem recovery alone

The experiences of so-called massive resections of the cerebral cortex which do not affect consciousness can throw little light on the problem of unconsciousness, because in most cases a relatively small amount of the brain tissue is removed and the integration

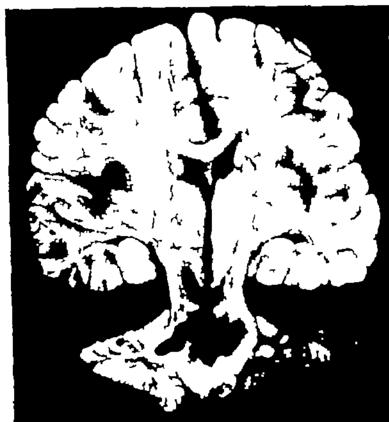


FIG 50

Massive hæmorrhages in the brain stem or pons of this type occasionally result from trauma.

of the rest of the brain function is left intact The danger of arguing from analogy can be illustrated in cases of excision of a lung Without one lung respiration can be carried on efficiently but it would be dangerous to assume that removal of the second lung would not interfere with respiration.

Moreover, I believe that there is no basic or uniform histological picture on which variegated contusional lesions may be superimposed that will account for the phenomena of concussion A so-called physiological injury is probably but a *minor degree* of the kind in which structural change can be demonstrated and presumably both physiological and organic neuronal injuries

occur in different parts of the same injured brain. In the cases where physiological injury is the dominant feature, recovery is probably rapid, whereas it is relatively slow when an organic injury predominates. In both types depth of unconsciousness is dependent on the number of neurones paralysed. A force sufficient to interfere with the vascular mechanism of the brain is unlikely to leave intact the more delicate neurones, and on evidence so far available it is difficult to believe that traumatic unconsciousness is ever produced by circulatory changes alone. On the other hand, circulatory disturbances often intensify the effects of direct neuronal injury whether of the organic or physiological type and may render permanent what otherwise may have been a reversible injury.

Summary of Concussion.—To summarise, I believe there are innumerable varieties of concussion and that they result from innumerable degrees and distributions of neuronal damage. From the clinical point of view there are innumerable varieties of unconsciousness due to innumerable combinations of faults in nervous integration. For clinical purposes deep coma may be regarded as a sign of brain stem injury, semicoma as a sign of bilateral hemispheral paralysis and confusion as impairment of function of the frontal lobes of the brain Consciousness, I believe, is the sum of general nervous integration and is not the function of any specific area in the brain, although, of course, many nervous pathways may congregate at some focal area where a small lesion can cause widespread interference with nervous function. Unconsciousness is brought about when a quorum of integration is absent, but here it must be clearly realised that what constitutes such a minimum necessity has not yet been discovered.

Neuronal injuries of submicroscopical dimensions, I believe, must vary in degree and in severity, as do macroscopical injuries. Therefore they probably lead not only to the paralysis and hyperactivity of the cell concerned but to any degree of intervening dysfunction, and in this way colour the final clinical picture. Indeed, so-called physiological injuries apparently can be progressive and lead to death without the development of secondary or epiphenomena (Jefferson). It has been suggested that further light will be thrown on the underlying neuronal change in concussion when measurements can be made of the electro-chemical metabolism of a damaged nerve cell. My belief is that the problem of concussion is essentially an anatomical one and will be solved by advances in microscopy. It will be solved when we can get a three-dimensional view of units of brain tissue which have not been subjected to processes (hardening, dehydration, staining and cutting) which inflict more damage on the tissues than the injury itself.

SECONDARY PATHOLOGICAL MANIFESTATIONS

The degree and extent of a primary injury is determined at the moment of impact, and if that part of the brain which is essential to life has not been destroyed by contusion, laceration or diffuse neuronal injury, the natural tendency of the patient will be towards recovery if other factors do not supervene. Unfortunately, secondary developments are invariable and often render fatal what otherwise would have been a reversible injury.

Shock.—Primary shock, according to a report issued by the Medical Research Council,[1] is a condition of collapse which may follow soon after the receipt of an injury and which is not due to hæmorrhage. It commonly occurs in acute cerebral trauma and usually responds rapidly to warmth, rest and relief of pain. Its underlying physiopathological cause is not known. Probably in cerebral trauma it is due to vasomotor paralysis of central origin and is part of the phenomena of concussion.

Secondary shock is a condition of circulatory failure. It develops insidiously some hours after injury and may be induced by cold, pain, hæmorrhage or toxins. Exposure in road accidents or after air raids is not very common, as the injured are rapidly cared for and transferred to hospital. It is on the battlefield and on the mountains where the factor of cold is important. The combination of chilling and severe concussion is almost invariably fatal. In climbing accidents there would be fewer fatalities if the patient were thoroughly warmed in a small tent or by any other means before an attempt was made to carry him down the mountainside. Pain and discomfort are common in the early stages of acute cerebral trauma. Often they are due to an irritative meningitis consequent on a subarachnoid hæmorrhage. Pain not only causes shock directly but also induces restlessness leading to exhaustion and to aggravation of any condition of collapse. Loss of blood in so-called 'closed injuries' is often considerable and even small hæmorrhages may produce adverse effects in those cases where the cerebral circulation is already embarrassed by other conditions. Profuse bleeding may occur from the nose, ears, cuts on the scalp, or from wounds elsewhere in the body, or into the subgaleal and subarachnoid spaces. Severe bruising of the face and crushing wounds of the limbs and body are often associated with states of concussion, and if it is agreed that absorption of broken-down tissue protein can cause secondary shock then toxæmia may often be responsible for this state in acute head injuries.

[1] Medical Research Council. "The Treatment of Wound Shock." M.R.C. War Memorandum No. 1. H.M. Stationery Office. London, 1940.

Whatever may be the precipitating factors of secondary shock the essential cause is diminished blood volume, with a consequent fall in heart output and blood-flow through the tissues of the body. The loss of blood volume in hæmorrhage is obvious. In other cases it is due to loss of plasma into the tissue spaces of the body, and thus is dependent on stasis of capillary circulation and on abnormal permeability of the capillary walls. As the arterioles are contracted some kind of vasomotor tone must be present, and thus the underlying sequence of events which leads to secondary shock in non-hæmorrhagic cases commences at the stage of capillary paralysis.

In a conscious patient secondary shock is characterised by weakness, pallor, a rising pulse rate, a falling blood pressure, sweating, vomiting and intense thirst. Such a picture, of course, in an unconscious patient is very difficult to distinguish from that due to a failing cerebral circulation directly consequent on an injury to the brain. In other words, secondary shock and a deteriorating state of concussion are almost indistinguishable.

In a certain and important group of injuries, which will be described in further detail later, a patient may remain in a state of semicoma for over twenty-four hours without the depth of unconsciousness or the general neurological picture altering one way or another. The first sign of retrogression in such cases may be an increase in pulse rate and a fall of diastolic blood pressure. Some of these adverse changes are due to secondary shock, and lives may be saved by an early blood transfusion

As regards prognosis, a reduction of the blood volume by 25 per cent. produces few symptoms, whereas a further reduction of 25 per cent. is serious. A pulse rate below 110 and a systolic pressure above 95 mm. Hg. associated with a blood volume of 75 per cent. are good signs, but figures worse than these are evidence of very grave deterioration.

MASSIVE HÆMORRHAGES

Extradural Hæmorrhages.—Extradural hæmorrhages large enough to be of surgical significance are extremely rare and are found in about 3 per cent. of cases in any large series of acute cerebral trauma.

They are, of course, extremely important, and it must be realised that the bleeding may come not only from the middle meningeal vessels but also from the diploic veins or dural venous sinuses (Fig. 52). Occasionally they may be associated with subarachnoid hæmorrhages or intrinsic damage to the brain.

Sinus Bleeding.—Tears in the walls of the dural venous sinuses are nearly always associated with overlying fractures. Fortunately they are rare, and this is due no doubt to the fact that the large dural channels, with the exception of the descending limbs of the lateral sinuses, are not embedded in deep bony grooves but run in contact with the flat surfaces of the skull and so are not necessarily lacerated when the overlying bone is fractured. As the sinus walls are fibrous and rigid they do not collapse or contract when torn as arteries do, with the result that bleeding is often profuse in spite of low venous pressure, and may be so rapid



FIG. 51

Even a small extradural hæmorrhage can cause a significant distortion of the brain and aggravate the effects of an intrinsic injury. Indeed it may be the deciding factor in the cause of death.

that the extravasated blood has not time to clot before the patient succumbs. Bleeding, however, is not invariably fatal, because the spicule of bone which lacerates the sinus may plug the opening into it or, as occasionally happens, the hæmorrhage may be limited by an adherent dura. Intrasinus pressure, normally low, is greatly increased by the slightest obstruction to respiration or by compression of the veins of the neck by tight clothing or bandaging. Any increase in venous pressure results in commensurate increase in bleeding.

Usually a tear takes the form of a small triangular flap and less commonly of a transverse or longitudinal split. Complete transections occasionally happen in open wounds, but in closed head injuries they are almost unknown and in both cases are invariably fatal.

In view of the frequency with which basal fractures converge on the pituitary fossa, injuries of the cavernous sinus are surprisingly rare, the superior longitudinal and lateral sinuses being those most commonly affected.

Diploic Bleeding.—Probably diploic bleeding is the most frequent cause of extradural hæmorrhage. Often not more than

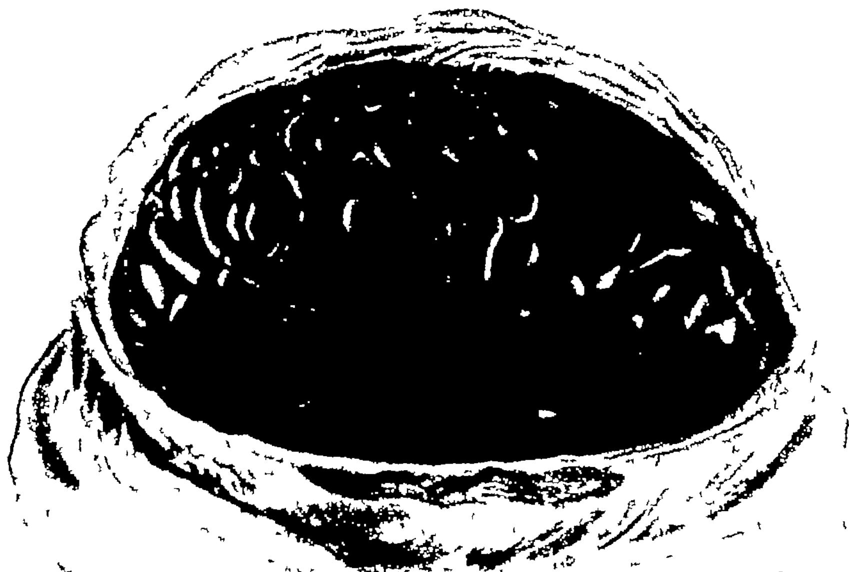


Fig 52

An extradural clot due to rupture of the middle meningeal vessels complicated by a subarachnoid hæmorrhage Such complications often explain why operations on extradural hæmorrhages are occasionally unsuccessful

a thin layer of blood collects; as this does not cause symptoms of cerebral compression it passes unrecognised and is absorbed by natural processes. Profuse diploic bleeding usually comes from numerous and widespread points, the precise localisation of which is impossible on clinical grounds since neurological signs point to the position of the clot or accumulation of blood and not to the origin of the bleeding which may be, and often is, some distance away. Radiology certainly shows the position and length of fracture lines from which the bleeding may emanate, but as these are often multiple and extensive, X-rays can rarely give information of more than lateralising value. The main difficulty in treatment is that the bleeding points may not be accessible through the exposure necessary for the evacuation of the clot or pool of blood which is compressing the brain

Middle Meningeal Hæmorrhages[1,3]—The middle meningeal artery arises from the internal maxillary branch of the external carotid artery, and enters the middle fossa through the foramen spinosum which lies just behind and lateral to the third division of the trigeminal nerve. Throughout its intracranial course it is accompanied by two venæ comites, which means that whenever the meningeal vessels are ruptured, bleeding takes place from both ends[3]. From the foramen spinosum the artery runs forwards and outwards on the base of the skull towards the tip of the great wing of the sphenoid bone, where it divides into an anterior and posterior branch. The anterior branch continues upwards and forwards to the antero-inferior angle of the parietal bone, which it deeply grooves or tunnels, and then turns upwards and backwards towards the vertex The posterior branch runs backwards and upwards across the squama of the temporal bone to the occipital region, this being the branch most commonly exposed in temporal decompressions

A point of interest is that the grooves in the bone caused by the anterior branches are commonly visible in X rays and may be confused with fracture lines Apart from its named branches, which are of little surgical importance, numerous small twigs run into the bone and are very easily avulsed whenever the dura moves away from the bone as the skull is deformed Inosculations across the middle lines between the vessels of either side are not very free, but they are widespread between the anterior and posterior branches on each side

The vessels may be ruptured in many ways They may be transfixed by a spicule of bone or lacerated by the edge of a fracture or torn by stretching as described in the previous chapter Bleeding may not take place immediately the vessels are torn, owing to the influences of shock, but as the circulation recovers and the blood pressure rises, rapid and profuse bleeding may occur The latent interval which so commonly occurs in middle meningeal hæmorrhages is not always due to shock but often can be accounted for by the ability of the brain to accommodate itself to a slowly expanding lesion for a long time before showing signs of compression In many cases the vessel bleeds from the moment the injury is inflicted, but often at a slow rate, due partly to incomplete rupture and partly to the resistance of the dura to stripping Immediate prodromal symptoms such as headache are the rule rather than the exception, and the rapid

[1] McKenzie K. G "Extradural Hæmorrhage" *Brit Jour Surg.*, 1939, 26, 346.
[2] Jacobson, W H. A "Middle Meningeal Hæmorrhage" *Guy's Hospital Report* 1885-86, 43, 147
[3] Wood Jones, F "The Vascular Lesion in Some Cases of Middle Meningeal Hæmorrhage" *Lancet* July 1912, 1, 7

In view of the frequency with which basal fractures converge on the pituitary fossa, injuries of the cavernous sinus are surprisingly rare, the superior longitudinal and lateral sinuses being those most commonly affected.

Diploic Bleeding.—Probably diploic bleeding is the most frequent cause of extradural hæmorrhage Often not more than

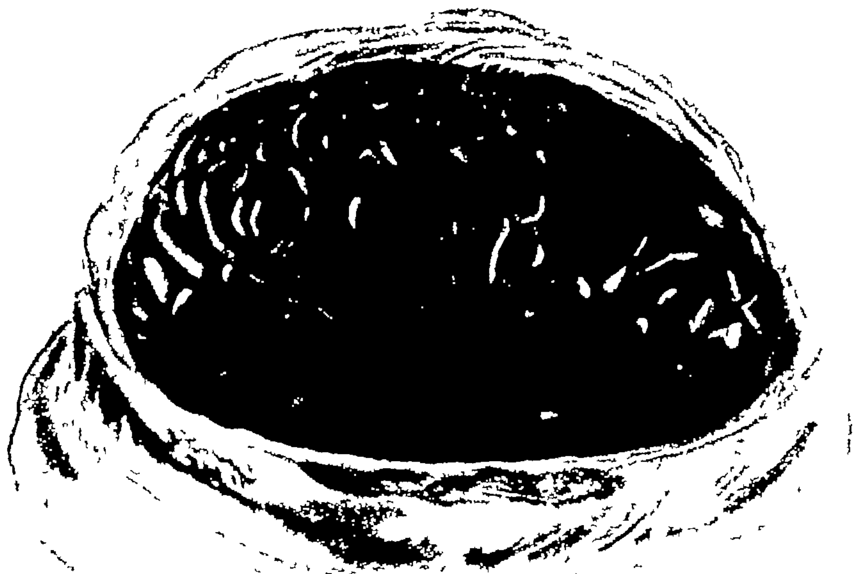


FIG 52

An extradural clot due to rupture of the middle meningeal vessels complicated by a subarachnoid hæmorrhage Such complications often explain why operations on extradural hæmorrhages are occasionally unsuccessful

a thin layer of blood collects; as this does not cause symptoms of cerebral compression it passes unrecognised and is absorbed by natural processes. Profuse diploic bleeding usually comes from numerous and widespread points, the precise localisation of which is impossible on clinical grounds since neurological signs point to the position of the clot or accumulation of blood and not to the origin of the bleeding which may be, and often is, some distance away. Radiology certainly shows the position and length of fracture lines from which the bleeding may emanate, but as these are often multiple and extensive, X-rays can rarely give information of more than lateralising value. The main difficulty in treatment is that the bleeding points may not be accessible through the exposure necessary for the evacuation of the clot or pool of blood which is compressing the brain.

Subdural Hæmorrhages —The arachnoid and dural membranes normally lying in close contact with each other are readily separable and enclose a potential space which can be converted into an extensive cavity by a hæmorrhage or leakage of cerebrospinal fluid into it.

Acute Subdural Hæmatomata.[1]—Bleeding into the subdural space may come from a dural venous sinus or a cortical vein if the arachnoid has been torn or, as is most common, from the short communicating veins which drain the cortical vessels into



Fig. 54

An acute subdural hæmorrhage is commonly associated with a more serious lesion elsewhere; in this case basally

the sinuses (Fig 55) Usually the bleeding is slight, but occasionally is profuse and may lead to fatal compression within a few hours *Often it is bilateral, and the blood tends to trickle* to the most dependent part of the subdural space according to the position of the head, and is usually found in greatest bulk over the outer surfaces of the temporal lobes

Acute Subdural Hygromata[2,3]—Hygromata may be due to laceration of the arachnoid, which allows cerebrospinal fluid to escape into the subdural space When large collections of fluid accumulate they are probably due to the tear in the arachnoid acting as a one-way valve, which allows fluid into the subdural space but not out of it. An alternative theory is that subdural hygromata are the result of transudations into the subdural cavity,

[1] Munro, D "Craniocerebral Injuries, their Diagnosis and Treatment" Oxford, 1938.

[2] Naffziger H. C. "Subdural Fluid Accumulations following Head Injury" *J A M A.*, 1924 82, 1751

[3] Hardman J "Asymmetry of the Skull in relation to Subdural Collections of Fluid." *Brit Jour Pathology* 1930, 12, 433-461

due to injury of the lining cells, and this theory is supported by the fact that the protein content of the fluid is often much higher than that of the normal cerebrospinal fluid.

Chronic Subdural Hæmatomata.—Chronic subdural hæmatomata have aroused widespread interest, not because of their frequency, which in fact is less than 1 per cent. in any clinical series of acute cerebral trauma, but rather because of the problems of their development and, to a lesser extent, on account of the excellent results of treatment by drainage.

The condition "pachymeningitis hæmorrhagica interna" has been recognised at least since the middle of the last century and

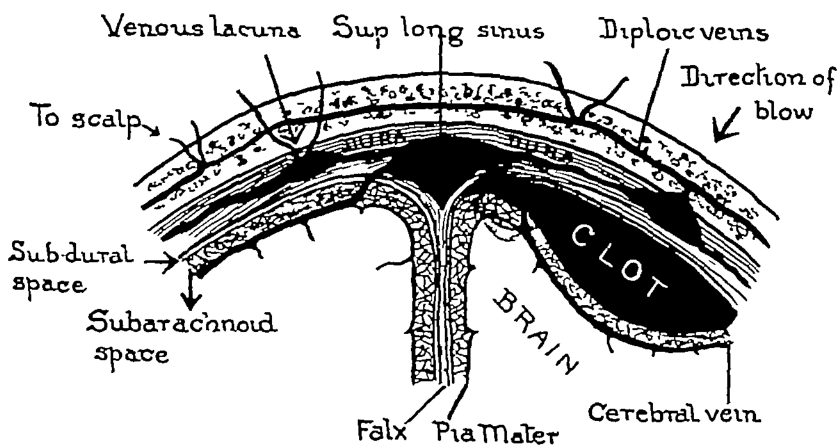


FIG 55

The tributaries of the parasagittal sinus The communicating veins which run from the cortical vessels into the sinus are commonly ruptured when the brain moves across the face of the dura In this case a unilateral subdural hæmatoma is shown

was fully described by Virchow [1] under the term "hæmatoma duræ matris." None the less, it remained for Trotter [2] to stress the hæmatoma's traumatic origin and to show that it often occurs after a very slight knock on the head.

The course of events in the development of any chronic subdural hæmatoma is complicated.[3, 4] A vessel, probably a vein, ruptures and then seals and the resulting hæmatoma becomes enveloped in a fibrinous membrane which later is organised by mesothelial invasion. The part of the membrane nearest the arachnoid remains thin and non-adherent, whereas the outer part thickens and becomes attached to the dura and is impossible to strip without rupturing numerous small blood vessels, which

[1] Virchow, R *Verhandl d phys-med Gesellsch*, 1857, 7, 134
[2] Trotter, W "Chronic Subdural Hæmorrhage of Traumatic Origin and its relation to Pachymeningitis Hæmorrhagica Interna" *Brit Jour Surg*, 1914, 2, 271-291
[3] Martin, J P "Chronic Subdural Hæmatoma" *Proc Roy Soc Med*, 1930 31, 24, 585 590
[4] Miller, C R "Chronic Subdural Hæmorrhage" *Med Bull Veterans' Adm*, 11, 332-336 Washington, 1934-35

is a point of some surgical importance At some phase in its life-history the encapsulated clot begins to swell, but why this happens is not known It has been suggested that small repeated bleedings could account for it, and this view is supported by the fact that at autopsy cerebral veins have been found either attached to the capsule or thrombosed within the clot. Putnam[1,2] showed that the adventitious membrane enveloping the clot becomes highly vascularised, and he suggested that oozings from the newly formed vessels would account for any increase in size of the hæmatoma In favour of this theory is the common finding of fresh blood within the clot, even after a considerable period has elapsed since the injury A popular and attractive theory is that of Gardner[3] who proved that the membrane of the hæmatoma possesses semipermeable properties He therefore offered the explanation that, as the clot disintegrates, its molecular concentration will increase and cause a rise in osmotic pressure with the result that cerebrospinal fluid will be drawn through the semipermeable membrane by the processes of dialysis[7] In favour of this hypothesis are the varied contents which may be found in the hæmatoma. These may be partly solid and partly fluid, or they may be wholly fluid Also the fluid may be black and viscous or it may be light brown and watery Furthermore, Munro has shown that the protein content of the fluid obtained from these hæmatomata actually increases as they break down On those occasions when the hæmatoma is not drained or removed by dissection it may fibrose or even calcify[4]

Subarachnoid Bleeding—Bleeding into the subarachnoid space is the most important and by far the most common form of massive bleeding due to acute cerebral trauma (Fig 56) In those patients who have been unconscious for over one hour, it is found in at least 75 per cent. of cases, and according to Nielson and Rand[5] the figure is as high as 85 per cent. The bleeding may come from any vessel on any surface of the brain In profuse hæmorrhages the communicating veins draining into the dural sinuses have usually been torn. The blood collects chiefly in the sulci, which it appears to fill, and also mixes freely with the cerebrospinal fluid which prevents its clotting Thus it is carried to all parts of the cerebral and spinal subarachnoid spaces and cisterns

[1] Putnam, T J., and Cushing, H. "Chronic Subdural Hæmatoma." *Arch. Surg.*, 1925, 11, 329
[2] Putnam, T J., and Putnam, I K. "Experimental Study of Pachymeningitis Hæmorrhagica." *Jour Nerv and Ment. Dis.*, 1927 65, 260
[3] Gardner W J "Traumatic Subdural Hæmatoma, with particular reference to the Latent Interval." *Arch. Neur and Psych.*, 1932, 27 847-858.
[4] Schüller A. "Hæmatoma duræ matris ossificans. *Fortschr a.d Geb d. Röntgenstrahlen* 1933, 51, 119
[5] Nielson, J M., and Rand, C. W "Fracture of the Skull: Analysis of One Hundred and Seventy-one Proved Cases: Diagnosis and Treatment of Associated Brain Injury" *Arch Surg.*, 1925, 11, 434-448.

Extensive intracranial clotting may occur when a large vessel is severed, but such occurrences are usually rapidly fatal.

According to Symonds,[1] the distinctive features of the cerebrospinal fluid taken at lumbar puncture are :—

(i) An even admixture of the blood which is the same in a series of specimens collected at the same puncture.
(ii) Absence of coagulation.
(iii) Pink, brown or yellow coloration of the clear supernatant fluid when the red cells have been allowed to sink to the bottom of the tube.

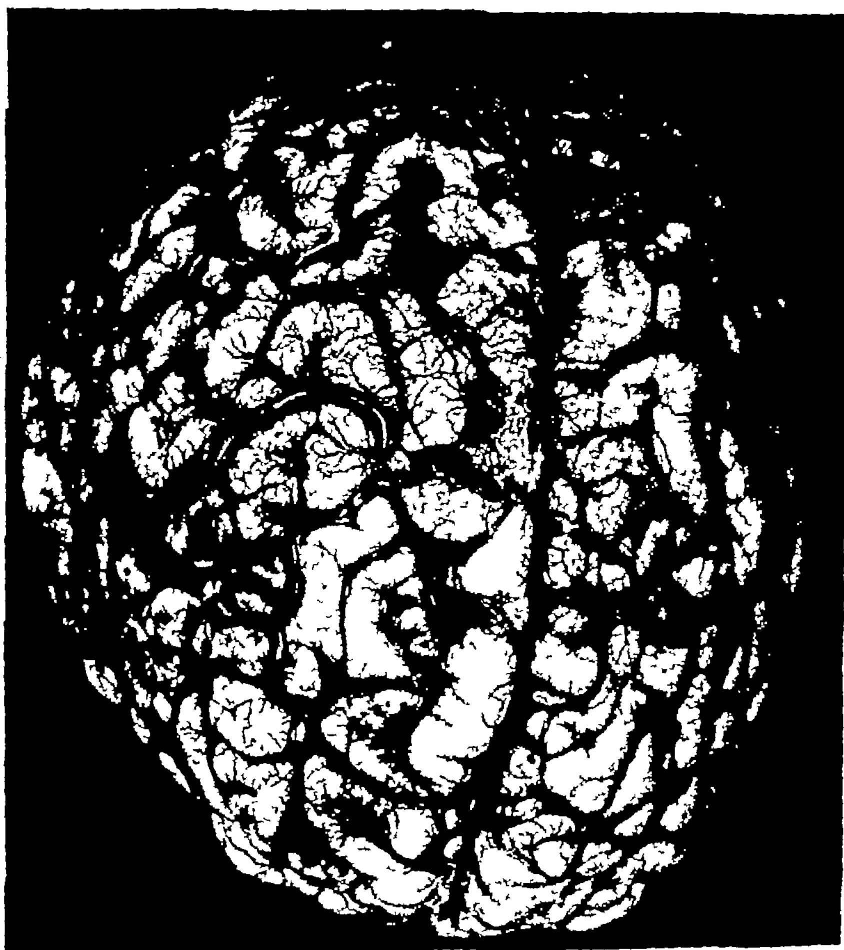


FIG 56
A mild subarachnoid hæmorrhage

Extravasated blood is rapidly hæmolysed and the products removed via the arachnoid villi, but xanthochromic staining of the cerebrospinal fluid may persist for many weeks after all active

[1] Symonds, C P "Spontaneous Subarachnoid Hæmorrhage" *Proc Roy Soc Med*, 1924, 17, 39-52

bleeding has ceased[1] The results of subarachnoid bleeding, when superimposed on the effects of the primary injury, are extremely serious. The irritation of the blood in the meninges, for instance, causes restlessness, with resulting cerebral congestion. This may cause increased bleeding or precipitate cerebral œdema. Blood loss may lead to a measurable degree of anæmia, and large quantities of extravasated blood have the compressive effect of a large clot. Also, resulting meningeal adhesions lead to faulty circulation or absorption of the cerebrospinal fluid

The amount of blood in the cerebrospinal fluid is of prognostic



Fig 57
A deeply placed intracerebral hæmorrhage resulting from trauma.

significance only within very wide limits, and more will be said on this subject in the following chapter

Subpial Bleeding.—The pia mater is firmly attached to the cortex of the brain and cannot be freely stripped, so that a subpial hæmorrhage always appears as a stain on the surface and never as a thick clot, although it may be so diffuse that it virtually constitutes a massive hæmorrhage. Usually it is an integral part of the contusion

Intracerebral Bleeding[2,3].—Large hæmorrhages deep in the tissues of the cerebrum (Fig 57), cerebellum or brain stem occasionally occur, and usually are due to pre-existing disease in the cerebral

[1] Bagley C. "Blood in Cerebrospinal Fluid: Experimental Data." *Arch. Surg.*, 1928, 17 18.
[2] Craig, W McK., and Adson, A. W "Spontaneous Intracerebral Hemorrhage: Etiology and Surgical Treatment with a Report on Nine Cases." *Arch. Neur and Psych.*, 1936, 35, 701
[3] Martland, H S., and Beling C. C "Traumatic Cerebral Hemorrhage" *Arch. Neur and Psych.*, 1929 22, 1001

vessels. On the other hand, large clots near the surface, the result of lacerations, are by no means uncommon.[1]

Intraventricular Hæmorrhages.—Profuse intraventricular hæmorrhages are commonest in children They are invariably fatal and are due either to tearing of the choroid plexus or to rupture of a large intracerebral hæmorrhage through the ventricular walls [2]

Œdema.—The term "cerebral œdema" implies that the brain is swollen, owing to an increase of fluid in the pericellular and



FIG 58

The honeycombed appearance, typical of cerebral œdema

perivascular spaces. It does not refer to the amount of cerebrospinal fluid in the ventricles or in the subarachnoid spaces, although it is probable that all the reservoirs of the brain act dependently. Also, it must be distinguished from "swelling of the brain," which is thought to be due to hydration of the cells themselves. Histologically, the picture of cerebral œdema is very typical; the tissues appear areolar or honeycombed, owing to the distension of the spaces mentioned above (Fig. 58). Œdema may develop early or late, and may be transient or persistent [3] [4] It may affect

[1] Friedman, E D "Massive Intracerebral Hæmorrhage of Traumatic Origin Injuries of the Skull, Brain and Spinal Cord" Baillière, Tindall & Cox London, 1943

[2] Hemsath, F A "Ventricular Cerebral Hæmorrhage in the New born" *Amer Jour Obst and Gyn*, 1934, **28**, 343

[3] White, J D, Brooks, J R, Goldthwait, J C, and Adams, R D "Changes in Brain Volume and Blood Content after Experimental Concussion" *Ann Surg*, 1943, **118**, 619

[4] Reid, W L "Cerebral Œdema" *Aust and N Z Jour Surg*, 1943, **13**, 11.

the whole brain or be confined to a lobe or, as is more usual, be localised around an area of contusion

Since the condition of œdema profoundly influences the mode of treatment in the acute stages of a closed head injury, it is important to inquire whether or not it occurs as often as is commonly supposed and to what extent it may embarrass the cerebral circulation or interfere with the nutrition of the neurones In America opinion has varied, many believing that œdema of a serious degree almost invariably accompanies a severe injury of the brain and often causes a rise in intracranial pressure with serious consequences [1-3] In Britain no definite conclusion has yet been reached on this point, but in neurological and neurosurgical circles it is believed that generalised œdema, as distinct from local œdema around a contusion, occurs much less frequently than has hitherto been supposed [4] My own opinion on the incidence of œdema is that the generalised form is rare whereas local swelling around a contusion is invariable. Furthermore, no one yet has been able to prove satisfactorily what part œdema plays in the production of unconsciousness or other neural dysfunction, and I believe it is not nearly so important a factor as subarachnoid hæmorrhage

That generalised œdema can occur there is no doubt. When operating under local anæsthesia, I have occasionally found the brain, in the acute phases of a head injury, under such great tension that it bulged into the wound as soon as the dura was opened, and later at autopsy have been able to show that the increased tension was not due to hydrocephalus and could not have been due to the amount of blood extravasated It cannot be objected that the increased tension found on surgical exploration was due to the anæsthetic, because local analgesia does not affect intracranial tension

The accumulation of the excess fluid in cerebral œdema probably is due to increased capillary permeability consequent either upon abnormal metabolites in the interstitial spaces, or on vasomotor paralysis in the region of the brain concerned When the condition of œdema has developed, venous congestion will tend to perpetuate it. In fact, a vicious circle results Œdema leads to increased intracranial tension, this causes a venous congestion, and venous congestion, by causing further capillary stasis, increases the œdema

Fay T "The Treatment of Acute and Chronic Cases of Cerebral Trauma by Methods of Dehydration." *Ann. Surg.*, 1935, 101, 76-13.

Lecount E. R., and Apfelbach, C. W "Pathologic Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries." *J A M A.*, 1920, 301

[3] Rand, C. W "Histologic Studies of the Brain in Cases of Fatal Injury to the Head. Preliminary Report" *Arch Surg.*, 1931 22, 738- 31.

[4] Greenfield J G Discussion on Cerebral Œdema. *Proc Roy Soc Med.*, 194-, 35, 5-5

Hydrocephalus.—Hydrocephalus is a state of dilatation of the cerebrospinal fluid spaces, and there are two main varieties—internal and external.

Internal hydrocephalus implies that the ventricular system only is affected. It is always caused by an obstruction, and this may be situated at the foramen or foramina of Monro, in the Sylvian aqueduct or at the foramina of Majendie and Luschka. According to the site of the obstruction, the ventricular system proximal to the block becomes dilated and thus one or all the ventricles may be involved.

As a complication in the acute stages of a cerebral injury internal hydrocephalus is rare, and when it does occur it is usually due to occlusion of the Sylvian aqueduct by a clot of blood seeping downwards from a lateral ventricular hæmorrhage. As a late sequel it is also rare, and in this case may be due to (1) stricture of the Sylvian aqueduct consequent on bruising of its walls, (2) adhesions in the posterior fossa, or (3) traumatic cysts compressing the fourth ventricle

External, or communicating, hydrocephalus implies that cerebrospinal fluid can escape from the ventricles on to the surface of the brain. It may be produced by (1) excessive secretion, (2) by faulty absorption, (3) by obstruction to the circulation of the cerebrospinal fluid or, as so often happens, by a combination of these factors Although it is impossible to state in percentages the incidence of external hydrocephalus, there is much clinical and post-mortem evidence to show that it is a happening of considerable frequency and importance in the acute phases of a cerebral injury. In decompressions performed under local anæsthesia, or when an inspection hole has been cut to establish a diagnosis, the bruised or normal cortex of the brain is often seen pushed away from the dura and not pressed tightly against it Also, on making a small incision through the dura and arachnoid, a stream of yellowish or lightly blood-stained fluid will often spurt out under great pressure to a height of several inches

In three cases of delayed hemiplegia—one examined at autopsy and two at surgical exploration—I have been able to prove beyond all doubt that the underlying pathological cause of the paralysis was an extensive unilateral external hydrocephalus.

The cause of external hydrocephalus is usually assumed to be faulty absorption of cerebrospinal fluid owing to blockage of the arachnoid villi by extravasated red blood corpuscles, and this explanation seems reasonable, since certain cells of the villous tufts are known to resemble those of the reticulo-endothelial system in their capacity to engulf and destroy foreign bodies.

Alternatively, Rand and Courville,[1] basing their views on a series of sixty-one autopsy specimens in which they demonstrated all degrees of injury to the epithelium of the choroid plexuses, concluded that such trauma would lead to excessive secretion of cerebrospinal fluid and to traumatic hydrocephalus.

Although blocking of the arachnoid villi and increased secretion of cerebrospinal fluid probably are the two main causes of any accumulation of excess fluid under tension in the subarachnoid spaces, interference with the circulation of the fluid is a causative factor which also must be taken into account, particularly since blood acts as a strong irritant to the leptomeninges and rapidly produces adhesions That surface anomalies of cerebrospinal fluid circulation can produce hydrocephalus was proved by my findings in one case at post mortem. These were as follows the left basal cisterns were blocked with a large clot of blood and the cortex of the brain on this side was firmly pressed against the dura, whereas on the right side the basal cisterns were free and the cortex covered by a deep external hydrocephalus Therefore, as there were no signs of bleeding on the left side, and as the arachnoid villi could not have been plugged with blood cells, it may be assumed that the obliteration of the cerebrospinal fluid spaces on the left side led to the hydrocephalus on the right side of the brain (Fig 59)

Herniations of the Brain.—Neurological signs in head injuries are produced in a diversity of ways They may be caused by (1) direct injury to the neurones, (2) by anæmia, local and general, (3) by circulatory anomalies of the cerebrospinal fluid, (4) by the strangulation of structures, over dural edges or taut vessels, or (5) by foraminal herniations It is with foraminal herniations that we are particularly concerned in this section

Whenever the brain swells or whenever the cranial space is encroached upon by an expanding lesion, such as an extradural hæmorrhage cerebrospinal fluid is forced into the spinal theca. Later, the basal cisterns are obliterated and the ventricles become flattened If the forces of compression or of œdema continue to act beyond the limits within which compensation is possible, by further expulsion of cerebrospinal fluid or of blood from the cranial cavity, processes of brain tissue are apt to be forced through the openings of the hiatus tentorii or of the foramen magnum as elongated herniations

The Tentorial Pressure Cone.—The tentorial pressure cone

[1] Rand, C. W., and Courville C. B. "Histologic Studies of the Brain in Cases of Fatal Injury to the Head: (2) Changes in the Choroid Plexus and Ependyma." *Arch. Surg.*, 1931 23, 357-425.

is a most important complication, and although it has been known for some time, it was so named by Jefferson,[1] who not only described its mechanics but illustrated its surgical significance.

During some phase in the rise of supratentorial pressure a process of brain tissue from the under and inner surface of the temporal lobe (uncus) herniates through the opening of the tentorium. The midbrain is displaced and compressed against the opposite free edge of the tentorium or, if the herniation is

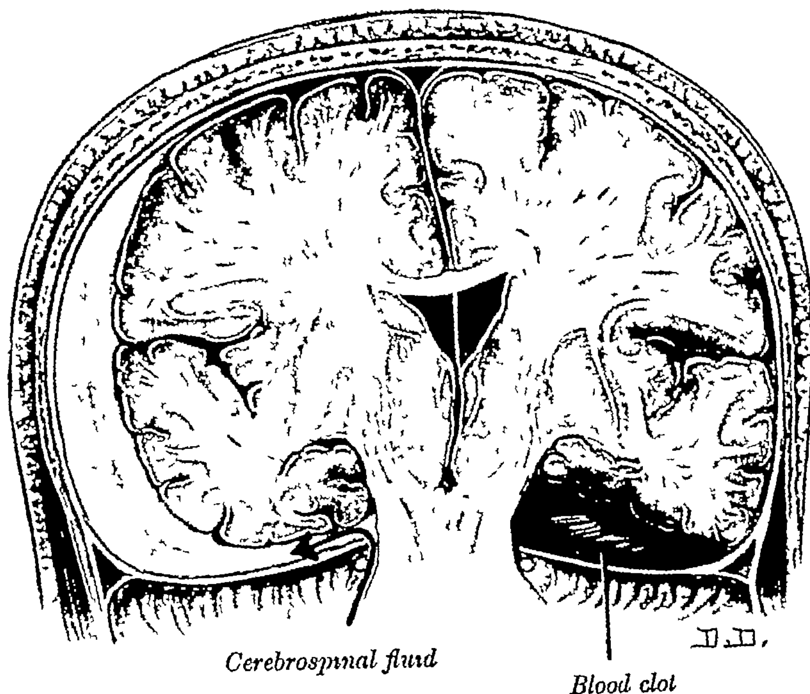


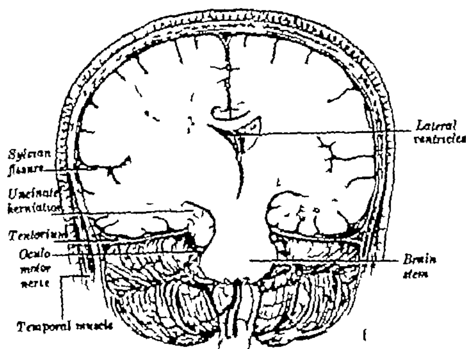
FIG 59
An external hydrocephalus, the result of a traumatic basal hæmorrhage

bilateral, between the two herniated processes. Conduction of impulses from the cerebrum is impaired, and the neural mechanisms below the compression are released from the control of influences of the higher centres. A primitive postural state known as decerebrate rigidity develops which is well known from animal experiment and follows transection of the brain stem just below the red nucleus. The muscles of the limbs go into a state of extreme hypertonus, usually in a position of extension but occasionally in flexion.

Another important complication is that the third nerve may

[1] Jefferson, G "Tentorial Pressure Cone" *Arch Neur and Psych*, 1938, 40, 857-876.

be stretched or compressed by the herniation It is this mechanism that accounts for a most important diagnostic sign, viz, the



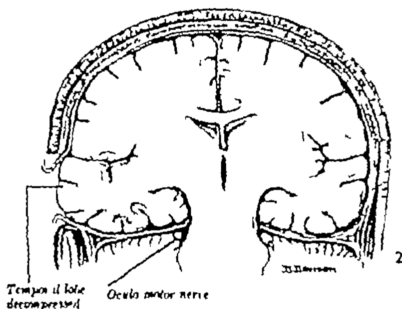


Fig 60

A tentorial pressure cone due to oedema, relieved by subtemporal decompression. It is in this type of case that operative treatment gives the most spectacular results.

fixed dilated pupil If not relieved a tentorial compression soon leads to death in coma (Fig 60).

The Cerebellar Pressure Cone of Cushing.—A rise of pressure in the posterior fossa may force the tonsils of the cerebellum through the foramen magnum into the spinal canal with resulting compression of the medulla oblongata. Such compression leads to respiratory embarrassment which might go on to complete failure. Although the circulatory centres may continue to function for a time, sooner or later they also fail and the patient dies. Fits in which the patient goes into opisthotonos occasionally occur. Cerebellar herniation is less frequent than tentorial herniation because it is the cerebrum and not the cerebellum that usually bears the brunt of an injury.



Fig. 61

Before the advent of chemotherapy diffuse meningo-encephalitis resulting from internal compounding was a common cause of death in head injuries.

Meningitis and Encephalitis.—Bacteria may enter the intracranial cavity through the nose or ears and produce meningitis or encephalitis.[1,2] Abscesses of the brain, in the surgical sense, do not occur in the acute phases of head injury for the obvious reason that a capsule has not time to form before the major issues are settled. As compound fractures of the frontal bone are more common than compound petromastoid injuries, infections through the nose are more common than those through the ears. Meningitis and encephalitis are often due to mixed infections; streptococci, staphylococci, pneumococci and heterogeneous bacilli being the bacteria usually concerned.

[1] Macewen, W. "Pyogenic Diseases of the Brain and Spinal Cord," 333. James Maclehose & Sons, Glasgow, 1893.

[2] Linell, E. A., and Robinson, W. L. "Head Injuries and Meningitis." *Jour. Neur. and Psych.*, 1941, 4, 23.

Infection may develop within a few hours of an accident and run a very rapid course. Such an occurrence is not always as unfortunate as it appears at first sight, since it is usually associated with a severe contusion or laceration of the brain, and merely aggravates what in any case would have been a fatal injury. Alternatively, late meningitis may develop at any stage of the healing process and transform what otherwise would have been a reversible injury into a fatal one. In the early stages, bacteria can gain direct access from the air sinuses to the leptomeninges before natural processes have had time to close the meningeal space by adhesions. This explains why it is so dangerous for

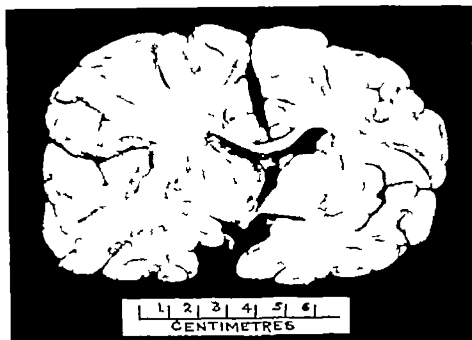


Fig. 63

Swelling of the right hemisphere of the brain, due to oedema.

a patient to raise his intracranial pressure by blowing his nose to remove an obstructing blood clot when a fracture of the anterior fossa is present. Infections are apt to be virulent, not only because the leptomeningeal spaces are widely open but because bruised brain tissue and extravasated blood form a very favourable medium for the proliferation of organisms.

Delayed meningitis in frontal injuries is often secondary to infections of those extravasations of blood which so commonly occur at the top of the nose either in the extradural space or in the sinuses themselves.

In petromastoid injuries delayed infection is more frequent than immediate meningitis and is due almost invariably to

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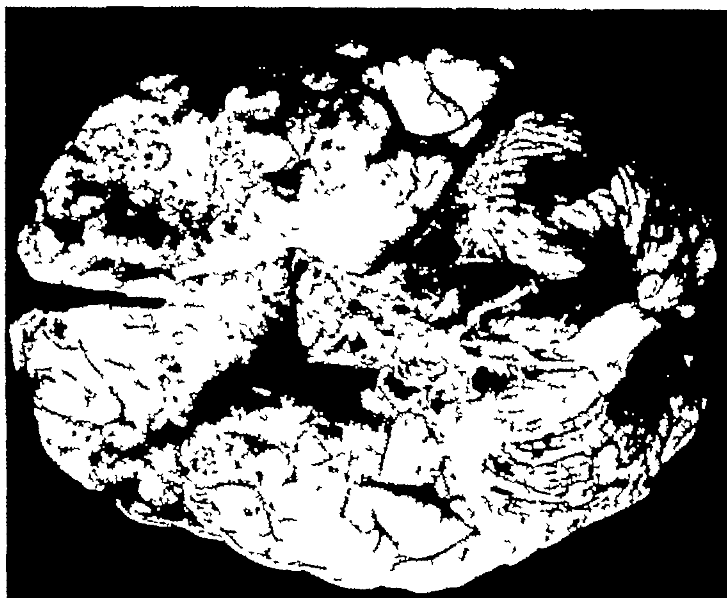


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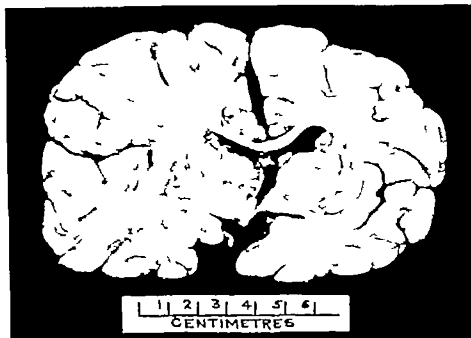


FIG 6*
Swelling of the right hemisphere of the brain, due to œdema.

a patient to raise his intracranial pressure by blowing his nose to remove an obstructing blood clot when a fracture of the anterior fossa is present. Infections are apt to be virulent not only because the leptomeningeal spaces are widely open but because bruised brain tissue and extravasated blood form a very favourable medium for the proliferation of organisms.

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suppurative otitis media which may or may not have been present before the injury. Naturally, pre-existing disease in the air sinus fractured is more likely to lead to either immediate or delayed infective complications, whether the infection comes by way of the nose or of the ears

Death in acute cerebral infections is due chiefly to malignant œdema (Fig 62) consequent on spreading encephalitis and not to the toxæmia of meningitis or blockage of the cerebrospinal fluid pathways by meningeal adhesions.

DEVELOPMENT OF THE NEUROLOGICAL PICTURE AND CAUSE OF DEATH

The neurological picture immediately following any violence to the head depends on one or a combination of the three primary types of injury. On this background further symptoms and signs are often rapidly added by secondary pathological developments Traumatic pathological states are essentially complex, and injuries to the same part of the brain may give rise to totally different signs according to whether they excite or paralyse the cells concerned. Complete loss of function in the motor cortex, for example, with paralysis of the corresponding limbs, will in itself cause no further change in the intracranial pathological state. On the other hand, hyperexcitability resulting in convulsive seizures will so raise venous, and thereby intracranial, pressure that further damage may be inflicted on the brain. This is also true of restlessness and delirium. The effects of straining on the volume of the brain and on the cerebral circulation have to be seen only once, when a bone flap is turned, for their importance to be appreciated. The brain bulges ominously; vessels become engorged and large veins may rupture and lead to serious hæmorrhage. Dysfunction, other than paralysis or hyperactivity, possibly plays a part in the production of the final clinical picture.

The results of contusion or laceration, apart from the secondary developments, depend on the part of the brain affected. It is the brain stem and basal ganglia that are essential to life. So long as the brain stem continues to function, respiration and circulation adequate for life will be maintained Hæmorrhages into the hypothalamus may destroy the controlling stations of the vegetative or autonomic system and cause metabolic crises, hyperthermia or acute perforation of the gastro-intestinal canal, any one of which may be fatal Contusions of the cerebral and cerebellar cortices produce neural deficits according to their position and extent, but they do not and cannot destroy life directly.

The extent and degree of primary types of injury are determined at the moment of the receipt of the violence and a static form of damage occurs Such damage may or may not be essentially lethal at the outset. When it is not, secondary developments may supervene and render fatal what otherwise would have been a non fatal contusion, laceration or diffuse neuronal injury

Secondary developments are progressive and not static pathological phenomena and, unlike the primary injuries which damage the neurones themselves, they produce their effects by raising the intracranial pressure and embarrassing the cerebral circulation Also, the chronology of their onset is variable, and each phenomenon by no means occurs in every case. Some degree of shock is usual, and in cases of severe concussion absence of subarachnoid bleeding is rare. Hydrocephalus is usually associated with surface hæmorrhages. It not only causes a rise in intracranial pressure but also may act as a local compressive lesion and give rise to localising signs, such as hemiplegia or aphasia. Œdema may be the only complication of a contusion When it remains local it is probably of no great significance. It may, however, spread rapidly to a whole hemisphere and dislocate the intracranial contents to such an extent that the functions of the brain become disorganised and death results It also causes all kinds of foraminal herniations Herniations may compress the brain stem at the foramen magnum or at the hiatus tentorii or they may compress vessels or nerves distant from the site of the primary injury It will be seen, therefore, that if a patient were co-operative after a severe head injury, the integration of the many pathological states which can affect the brain as the result of acute cerebral trauma is so complex that diagnosis and treatment would still remain a very difficult problem.

Patients moribund from the start who show no sign of mental or physical improvement in spite of treatment usually have received a primary type of injury to the brain which is essentially fatal Such patients usually die within twelve hours At autopsy, severe lacerations or extensive contusions are often found, and occasionally large areas of the brain are completely pulped Extensive surface hæmorrhages are often present, but, as far as can be judged, death would have resulted even if they had been absent. In this group of cases nothing useful can be done surgically

Death may occur within twelve hours from secondary developments alone and particularly from the compression of surface hæmorrhages In such cases a large vessel has usually been torn In my experience the vessels concerned in order of

To settle this problem I treated a hundred cases conservatively and a comparable series, as far as was possible, actively The results of this experiment were that fatalities were 30 per cent. higher in the conservatively treated group than in the other Therefore, in my opinion, not only can many lives be saved by skilled treatment, but sequels can be reduced to a minimum and morbidity rates considerably improved

AIR-RAID CASUALTIES

From the beginning of the present war up to November 1943, 50,263 people were killed and 162,485 injured, as the result of enemy action from the air In No 1 Region, which embraces the counties of Northumberland, Durham and North Yorkshire, with their long seaboards on the North Sea, and with the river valleys of the Tyne, Wear and Tees, many people have been killed and injured [1] During the years 1941, 1942 and 1943, 355 cases of injury to the head were reported to the Head Injury centre of this Region, many of these were seen and some treated by mobile teams The majority, however, were treated by the surgical staffs of the receiving hospitals. Of the more severely injured, 72 cases were transferred to the Head Injury Centre for treatment The details of these cases were as follows —

Concussion

Scalp intact (with or without fracture)	13
Scalp laceration (skull intact)	15
Compound fracture of skull (dura intact)	14
Compound fracture of skull (dura torn)	7
Perforating wound (foreign bodies in brain)	3

Injuries of Scalp and Skull without Concussion

Laceration of scalp	6
Closed fracture of skull	2

Delayed Cerebral Complications

Cerebral abscess	3
Osteomyelitis of skull	1
Intracerebral hæmorrhage	1
Anxiety neurosis	3

[1] The publication of these figures is not permitted for security reasons.

Fractures of Spine

With cord injury	2
Without cord injury	2

There were associated injuries in 27 of the above cases; these were:—

Fracture of long bones	4
Fracture of short bones	4
Fracture of ribs	1
Thoracic injury	1
Abdominal injury	2
Eye injury	3
Fracture of pelvis	2
Fracture of jaw	1
Burns	1
Extensive bruising	8

Of the above 72 cases, death occurred in 11, the causes being:—

Brain injury	3
Brain injury, complicated by infection	4
Cerebral embolism	1
Penetrating wound of spinal cord (with severance of cord in one case)	2
Multiple injuries	1

The outstanding features of casualties following severe air raids are:—

(*a*) The relatively large number of fatalities in relation to those injured.

(*b*) The variety in nature, in severity and in the combination of the injuries.

(*c*) The begrimed and blood-stained condition of the injured

These characteristics are, no doubt, due to the manner in which the injury is inflicted. As described in Chapter I, a victim may be injured directly by blast, in which case he may receive a relatively simple compressional injury or may be blown to bits, according to his proximity to the explosion. He may be struck by flying bomb fragments, which may be small or large and which may be travelling at their greatest speed or coming to rest. The body may be lacerated or pierced by small fragments of flying glass or wood set into rapid motion by blast or suction Injury may be inflicted by the forces of acceleration or deceleration, either of the rotational or linear type, for example, a man may be thrown and brought to rest either by the direct forces of blast

or may fall from an upper storey to the ground floor when his house is destroyed Finally, he may be crushed by falling masonry, suffocated, burnt or scalded

In the light of these possible modes of infliction of injury, let us consider some of the episodes gathered from personal experience

A group of nurses were sheltering in a cellar when a bomb exploded immediately above but did not break down the walls about them. There was a tremendous commotion, the whole room began to vibrate, some of the nurses were thrown off their balance and some thrown from their chairs The room became filled with dust and they felt as if they were going to suffocate The episode was soon over, and shortly afterwards they were talking together excitedly When the raid had passed, they came up into the light, gay and confident, but obviously relieved to find themselves safe. They were covered with dust, and small pieces of plaster were driven into the roots of their hair They needed a change of clothing, a bath and a sleep Many of them were able to go on duty the same day and carry out their work efficiently As far as could be judged, nothing had happened to these young women either physically or emotionally to do them any lasting harm

On another occasion an overwhelming tragedy occurred when a bomb pierced a large building and exploded in the basement. Of 192 people sheltering there, 105 were killed instantly Between 30 and 40 people received injuries which necessitated surgical treatment, of these 22 received injuries to the head, 7 of which were serious wounds, the dura being penetrated in 4 cases

Of the fatalities, I had the opportunity to examine some 30 cases, and found that nearly all of these had received severe and multiple injuries, each of which might have been fatal The main head wounds were large compound fractures of overwhelming magnitude with massive pieces of bone driven into the brain, in some cases the whole of the head was deformed The skin often was so blanched as to suggest that there had been a massive loss of blood rather than that the colour was due to shock. During the rescue, men, women and children were found conscious with their bodies or limbs pinned and crushed by heavy girders and stones. One woman was held fast and unable to move, while her baby of a few months lay injured close beside her This episode is mentioned to illustrate the fact that severe mental as well as physical shock may be inflicted at the time of injury

In this episode it is obvious that the occupants of the shelter had been subjected to all the possible modes of infliction of injury and had suffered accordingly

As an example of the direct effect of blast, several instances can be recalled of people being blown to bits, so that only minute fragments of their bodies could be discovered after the explosion. After one air raid people were carried into hospital retaining the exact position in which they were crouched immediately before being killed; in particular, they were flexed in the sitting position with their hands lifted up to their face; that this is what happened was vouched for by some of the people present in the shelter who, by some miracle, escaped with their lives. Moreover, it was obvious from external examination that many of the fatalities had not been thrown or struck by falling debris. These peculiar results were observed following a direct hit on a surface shelter which blew the walls and roof away, so that little debris fell on the injured. The dead presumably had been killed instantly by the blast. In some episodes the outstanding feature is burns, resulting from people being trapped in burning buildings.

Peppering of the skin and of the deeper parts of wounds with bits of mortar, brick and grit has been a remarkable feature in air-raid casualties. The face and exposed parts are blackened, stippled and blood-stained; the hair also is matted with dust or debris, and it is this begrimed condition which gives the injured their characteristic and rather frightening appearance (Fig. 63).

The peppering with grit is of importance in surgical procedure, as it often renders shaving of the scalp difficult when preparing the patient for operation. Scalp wounds, too, are apt to be extensively contaminated, necessitating careful exploration even of apparently simple cuts. Wide excision of the galea and periosteum has often to be done before a wound can be adequately cleaned, because the foreign matter is embedded firmly in the deep tissues and cannot easily be wiped, washed or brushed away.

Blast injuries of the lungs are by no means uncommon and complicate injuries of the head. In these cases a general anæsthetic should never be given for the treatment of cerebral wounds or, for that matter, for wounds elsewhere in the body.

Flying pieces of glass may lacerate large areas of the skin, body or scalp; they may destroy the eyes or even pierce the skull and lacerate the brain. Experience has also shown that small pieces of wood are dangerous missiles, and in Chapter I a case is quoted in which the brain was perforated through the orbit and sphenoidal fissure by a fragment of wood.

A child who had had a large compound depressed fracture of the skull excised came under my care because the scalp refused to heal as a result of persistent deep infection. On re-exploration of the wound it was found that a large piece of wood had skidded

so far between the dura and the bone that it was completely hidden and no part of it presented in the open wound

Physical shock plays its part in air raid casualties In general, however, its manifestations have been little different from those we are accustomed to see in the severely injured in peace time. With warmth, rest, drinks, reassurance and relief from pain, most of the injured have made a rapid and lasting recovery from primary shock Blood transfusions usually have been necessary in those cases where there has been a loss of blood, either from an



Fig. 63

Patient with "blast lungs some twenty four hours after a bomb injury Multiple small wounds over the whole of the face and scalp. (*Kindly lent by Surgeon Rear Admiral C P G Wakeley C.B.*)

external or internal hæmorrhage Death from anæmia can occur from subcutaneous rupture of a large vessel such as the gluteal, the blood tracking along interstitial spaces Such hæmorrhages can easily escape detection and the condition be mistaken for shock Secondary shock commonly follows crushing of the limbs and blast injuries to the lungs and abdomen In head injuries unaccompanied by severe damage elsewhere in the body, shock, either primary or secondary, has been no different from that seen in patients who have received their injuries in industrial or road accidents

In large shelters, good discipline and normal behaviour, with rare exceptions, has been most noticeable Certainly during raids there is usually a sense of anxiety and of excitement, but

none the less, people have subdued very successfully whatever degree of fear they were experiencing. Morale tends to deteriorate with repeated and prolonged bombing, but in my experience there have been no outbreaks even of minor panic

The demeanour of the injured has been exemplary, their fortitude amazing. Many in the acute stages have realised that their husbands, wives or children have been killed and their homes destroyed. In spite of this they have remained calm and apparently philosophical. They give the impression that the whole episode was an act of God rather than a holocaust caused by the agency of man. It has been realised that it is a bad psychological error to allow casualties of a previous night to see the newly injured of the following night; it is too poignant a reminder of what they have suffered.

At this point it would be fitting to mention the superb poise and unselfish devotion to duty of the nursing staffs during long hours of danger and strain. The high morale of hospital patients has been due, in great measure, to the nursing staffs' reassuring influence in moments of stress.

The cycle of emotional events in normal individuals appears to be as follows: During danger there is a natural sense of fear, allayed by activity and aggravated by inactivity or by anxiety for relatives or friends; this is followed by a sense of relief immediately the danger has passed.

No doubt it will often be asked in the future why air raids have so signally failed to produce a serious deterioration in morale. The more obvious reasons are the absence of sufficiently sustained bombing, a certain toughness in the national character, and speedy rescue and prompt medical services In my opinion, however, the main reasons are more fundamental or biological. It is useless to try and drive women from their homes and the things they cherish; thus, so long as morale remains high in women, so long will it be sustained in children and men.

A PATHOLOGICAL STUDY OF AN OVERWHELMINGLY SEVERE CLOSED INJURY OF THE HEAD



Fig 64
Bruising and grazing about the face and head.



Fig 65
Reflection of the scalp reveals a stellate fracture of the frontal bone.



Fig 66
A large extradural hæmorrhage in the temporal and occipital regions

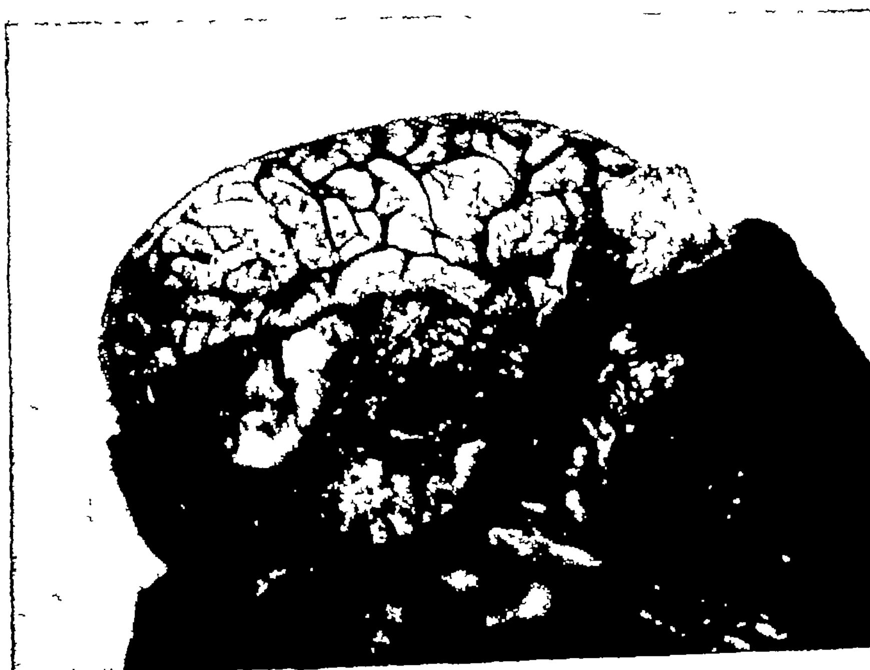


Fig 67
Removal of the dura reveals subarachnoid bleeding



FIG 68

Transverse section shows a bilateral extradural hæmatoma. The brain has fallen away from the frontal fossa by its own weight.

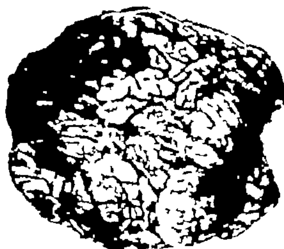


FIG 69

Bruising and laceration of the undersurface of the brain.

A PATHOLOGICAL STUDY OF A CASE WHERE THE INTRINSIC INJURY WAS NOT OVERWHELMINGLY SEVERE



Fig 73

With the scalp reflected forwards and backwards a fracture running along the line of the sagittal sinus is clearly depicted Note the subgaleal hæmorrhage, loss of blood into this compartment can be considerable, constituting a form of internal bleeding also it is in this layer that adhesions are so prone to give rise to headaches



Fig 74

Vault of skull removed The extradural bleeding is widely spread but not profuse



Fig. 75
Dura reflected; note diffuse subarachnoid bleeding.



Fig. 76
An excellent illustration of the rupture of a parasagittal vein. It is rupture of these veins that is so liable to cause subdural and subarachnoid bleeding.



Fig 77
Bruising on the under surface of the frontal and temporal poles of the brain



Fig. 78
Reflection of the dura shows a fracture of the base running into the paranasal air sinus



Fig. 79

In this case there was no macroscopical evidence of damage to the brain stem or to any part of the cerebellum.



Fig. 80

Superficial bruising at the base of the brain without frank damage to the hypothalamus.

Without the occurrence of secondary pathological phenomena, it is doubtful whether the patient would have succumbed.

AN ATLAS OF AUTOPSY FINDINGS IN CLOSED INJURIES OF THE HEAD



FIG 81

In this case the hemisphere on the side of the laceration was swollen with œdema There were no macroscopical lesions elsewhere

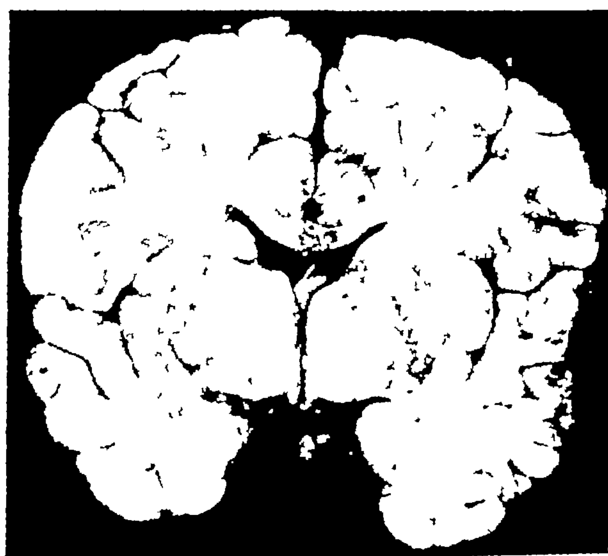


FIG 82

Hæmorrhages into the corpus callosum are by no means uncommon They probably also occur in non-fatal injuries What neurological sequels result is a matter of conjecture



FIG. 83
Occasionally even in very severe injuries the macroscopical damage is confined to one side of the brain. The intrinsic basal injury here is a severe one



FIG. 84
Displacement of the ventricular system resulting from a combination of laceration and subdural hemorrhage



Fig 85

Damage of the internal surface of a hemisphere against the falx cerebri is by no means a rare occurrence The large intracerebral hæmatoma in this case has resulted from the tearing of a large blood-vessel The flattening of the hemisphere has been caused by a massive local subdural hæmatoma

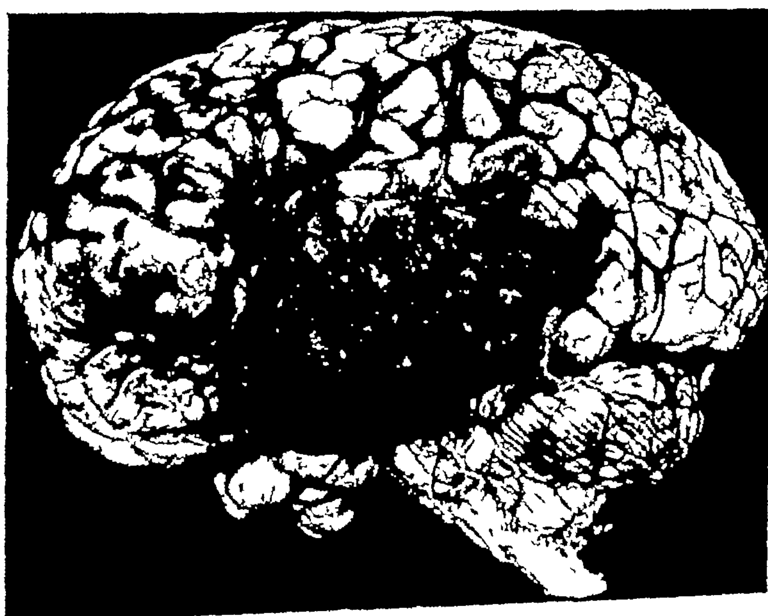


Fig. 86

An instance of a massive local subarachnoid hæmorrhage In this case there was no macroscopical evidence of basal intrinsic injury

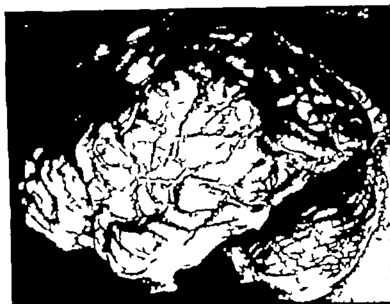


FIG. 87
Tentorial hæmatomata are difficult to diagnose and difficult to treat

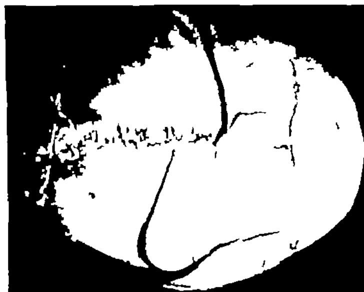


FIG. 88
Fractures of the skull in children are commonly of the massive type. Often they serve a natural decompressions and conserve life

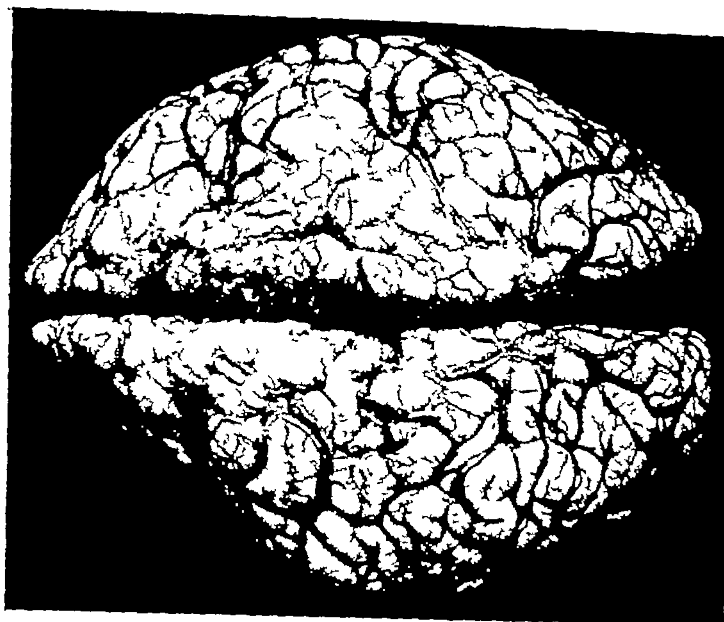


Fig 89

Shrinkage of the frontal pole of the brain associated with an acute subdural hæmorrhage Macroscopically there was no damage in the basal or brain stem areas in this case

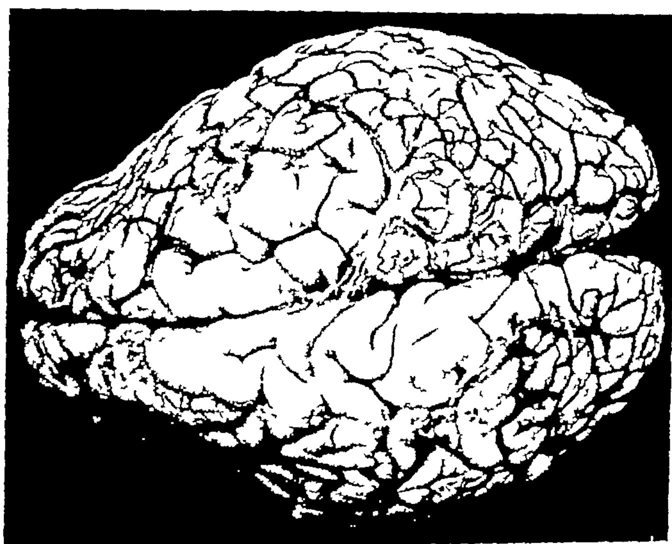


Fig 90

An example of shrinkage of the brain following a closed head injury On both sides there was a massive subdural collection of yellow-stained fluid of high protein content The specimen was taken from a patient aged 60 years, who before the accident was mentally sound. The W R was negative It is possible the condition found at post-mortem was an old standing one and was coincidental to the injury and not a result of it

CHAPTER III

DIAGNOSIS OF CLOSED INJURIES OF THE HEAD *

DIAGNOSIS[1-7] in acute cerebral trauma of the closed type is usually a difficult problem, since many of the patients resent examination or are profoundly unconscious Owing to lack of co-operation the customary routine investigation of the nervous system is impossible and much essential information has to be sought by indirect means Moreover, the neurological picture is often so confused that the whole brain seems to be affected, which, in fact, it is With reasonable care, however, a reliable impression of the nature of the primary brain injury can be obtained and secondary developments diagnosed at a stage when they are still amenable to surgical treatment.

Primary shock must always be treated before a detailed examination is made, not only in observation of surgical first principles but because no trustworthy neurological information can be obtained so long as this state exists. A few minutes only are necessary to ascertain the patient's general condition and to determine how deeply unconscious he is and whether or not there are associated injuries needing urgent treatment. Apart from careful scrutiny of the scalp and body for open wounds, detailed examination may be deferred for half an hour, so that the patient may be warmed up, as he is often admitted to hospital thoroughly chilled

Examination —Examination must be carried out in a warm room and under good lighting When these two conditions have been obtained, all the covering bedclothes should be removed

* The term "closed" means that the brain has not been penetrated by a missile and that a compound fracture of the vault has not occurred; a fracture of the base opening into the ears or nose may be present.

[1] Dott, N. M. "Thompson and Miles Manual of Surgery" 1939 2.

[2] Jefferson, G. "Discussion of the Diagnosis and Treatment in Acute Head Injuries." *Proc Roy Soc Med.*, 1932, 25, 742.

[3] Munro, D. "Craniocerebral Injuries; Their Diagnosis and Treatment" Oxford University Press, 1938, 412.

[4] Riddoch, G. "Discussion of the Diagnosis and Treatment of Acute Head Injuries." *Proc Roy Soc Med.*, 193., 25, 733.

[5] Russell, W. R. "Discussion of the Diagnosis and Treatment of Acute Head Injuries." *Proc Roy Soc. Med.*, 1932, 25, 751

[6] Symonds, C. P. "Concussion and Contusion of the Brain and their Sequelæ" "Brock's Injuries of the Skull, Brain and Spinal Cord" Baillière Tindall & Cox. London, 1943.
Trotter W. "Injuries of the Skull and Brain," "Choyce's System of Surgery" 3, 309 Cassell & Co. London, 1932.

and the patient completely stripped, otherwise it is often impossible to get a true perspective of the complex problem presented and important signs may be overlooked. First of all, spend a few minutes purely in inspection: analyse the posture of the patient and observe his spontaneous movements: watch his facial expression and note his colour and type of breathing. When this has been done, make sure that there are no fractures of the limbs which require temporary splinting to prevent them becoming complicated or compound if the patient is, or should become, restless. From the previous chapter it will have been learned that serious injuries to the chest, spine and abdomen often complicate cases of head injury Therefore the abdomen should be palpated for rigidity and percussed for the presence of free fluid or air in the peritoneal cavity, the latter being indicated by loss of liver dullness. The chest wall should be palpated for fractured ribs and the chest cavity percussed and auscultated for pneumothorax or hæmothorax Also, a finger should be run along the spine to detect the possible irregularity of a fracture dislocation [1]

If these things are not done a reliable estimate of prognosis is impossible and treatment of an injury elsewhere in the body may be omitted, which is just as important as that directed to the damaged cerebrum.

When associated injuries have been noted and their severity assessed, the head should then be examined for signs referable to injury of the skull, and such data should be kept separate from that referable to the brain, otherwise a jumble of confusing information will accrue.

The hair is often matted with blood, which must be washed cleanly away if the scalp is to be examined adequately and possible compound fractures disclosed. In any case, wounds of the scalp not accompanied by underlying fractures are important, because if not correctly treated they may lead to fatal or troublesome septic complications. The face should be cleaned and abrasions treated so as to minimise subsequent infection. Pools of blood must be dabbed from the ears, otherwise it is impossible to know whether the bleeding comes from a fractured base or has merely trickled into the external auditory meatus from a wound in the soft tissues Finally, attention should be directed to those signs which are referable to the cerebral injury, and this is by far the most difficult part of the examination and one which necessitates a great deal of practical experience as well as a knowledge of theoretical neurology Restlessness is never a sufficient reason for omitting to make a detailed examination.

[1] Walshe, F M R "Note on a Commonly Unrecognised Type of Injury to the Cervical Spine and Spinal Cord in association with Head Injuries" *Lancet*, August 5, 1944, 173

Signs referable to the Skull (Figs 91 and 92) —In recent years the importance of damage to the brain has so often been stressed that the older habit of devoting the main attention to the condition of the skull has tended to become overcorrected Although it is true that what happens to the brain and its covering membranes finally determines whether the patient lives or dies, it must be realised that information obtained from the skull can materially assist in the diagnosis of the nature of the cerebral injury Moreover, fractures of the paranasal or petromastoid regions, if not diagnosed and treated correctly, *lead to meningitis and encephalitis*

When death occurs within twelve hours the brain injury is usually associated with an extensive fracture of the base In cases in which the signs of injury to the brain are minimal, a fracture *of the skull indicates that a considerable force has been applied* to the head, and so suitable precautions may be taken, particularly by repeated observation It is unrecognised cases in this category that are sometimes allowed to go home from casualty departments, only to die from cerebral compression due to an extradural or subdural hæmorrhage When an extradural hæmorrhage is suspected but cannot be diagnosed with certainty on clinical signs alone, the evidence of a fracture line crossing the middle meningeal grove will influence a decision to make an early exploration Even in closed fractures a spicule of bone may pierce the dura mater and lead to traumatic epilepsy if not removed and the dura repaired

Clinical Signs —On no occasion must the diagnosis of a fracture of the skull be attempted by the elicitation of crepitus No useful information is likely to be gained by this method and serious damage may be done thereby A loose fragment of bone lying outside the dura may be driven inwards to lacerate the brain, or a hæmorrhage may be started which would not otherwise have happened

The edges of a large depressed fracture can be palpated with certainty but in small depressions a bony edge can be very closely *imitated by the indurated edges of a centrally fluctuating hæma* toma in the scalp and there are no reliable clinical means of distinguishing between the two Linear fractures unless they are widely open, cannot be diagnosed by palpation Extensive and boggy *swellings under the scalp indicative of large subgaleal* hæmorrhages always mean that the bone has been broken Thickening of the temporal muscle and postmastoid bruising are also reliable signs of fracture On the other hand, bleeding from the nose or ears often comes from laceration of soft tissues *Profuse* and persistent bleeding from these sources is suggestive of fracture, but the only incontrovertible evidence that the skull has been

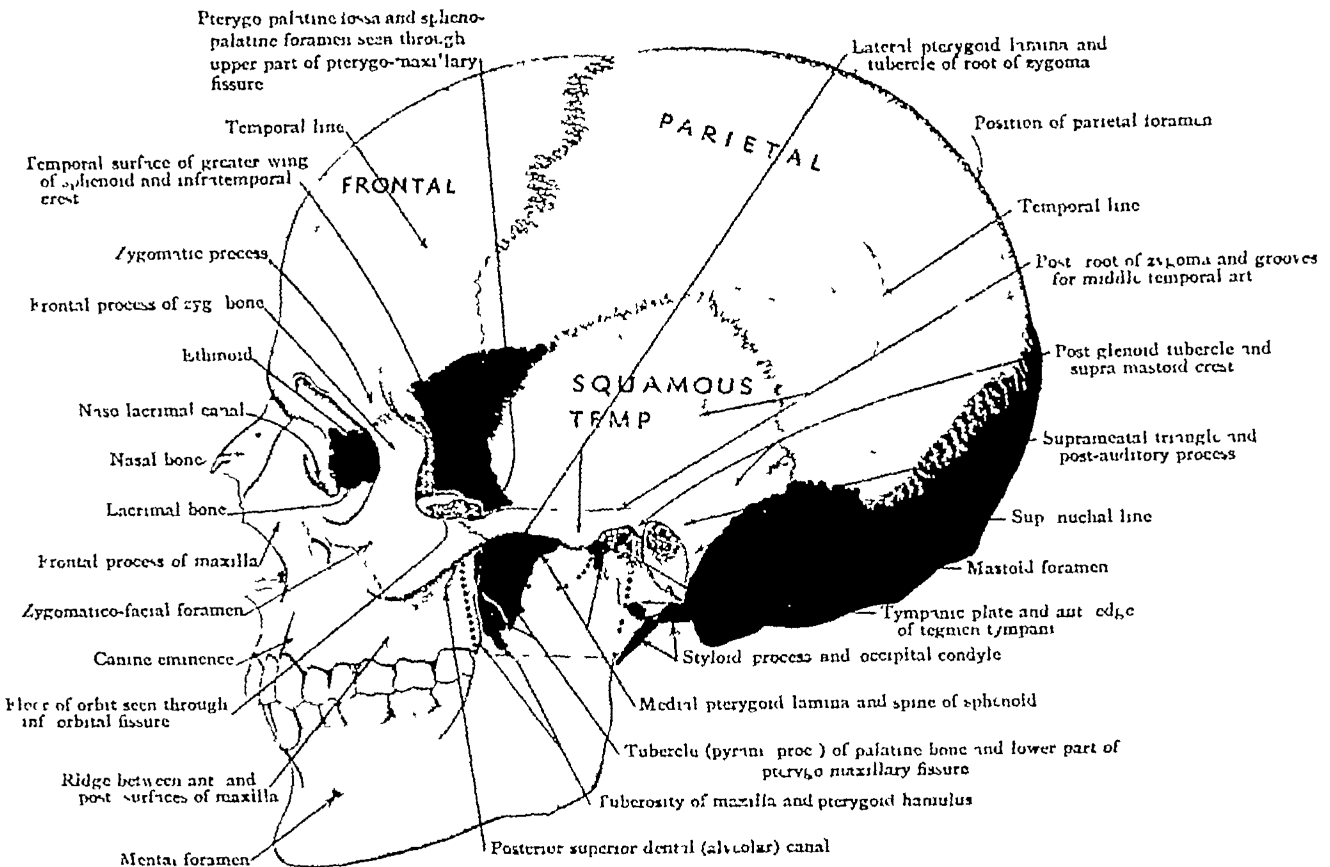


Fig 91
Side view of skull. (*E B Jameson*)

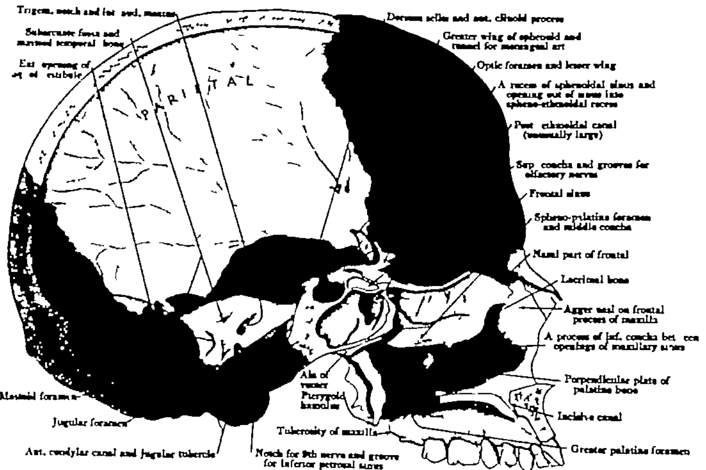


Fig. 92
Sagittal section of skull. (*E. B. Jamieson*)

broken is the presence of cerebrospinal fluid or brain tissue in the discharge.

Subconjunctival hæmorrhages are significant of fracture only (1) when they cause œdema of the conjunctiva, (2) when they are so extensive that it is impossible to see beyond their posterior limits in any position of the eye (Fig. 93), or (3) when they are so large that they displace the eyeball and restrict its movements Finer details than these of differentiation between the hæmorrhages of a fracture and a "black eye" are of little practical value since



(a) (b)

Fig 93

a, This type of intra-orbital hæmorrhage is indicative of fracture of the anterior fossa
b, A flame shaped hæmorrhage is caused by contusion of the soft tissues and not by fracture

a fracture of the base may not lead to an intra-orbital hæmorrhage, but may be and usually is associated with a black eye. Gross deformities of the skull are incompatible with life.

Radiography.—Since the nature of a cranial injury can aid in the diagnosis of the underlying cerebral state, radiography should be used as a routine measure in the acute stages of cerebral trauma. Usually it is best to take the photographs immediately at the end of the clinical examination, as clinical findings will determine the views that may be necessary. Unfortunately, the decision as to whether or not radiography should be used is often determined by convenience rather than by conviction, or by someone other than the surgeon, and these faults ought to be corrected. It is unwise further to increase shock by transporting the patient to the main X-ray department of the hospital for detailed examination, but there is no reason why he should not be examined in his bed by means of a portable apparatus. With reasonable care, satisfactory films can be obtained. Useless films are usually due to faulty exposure rather than to misbehaviour of the patient. With sufficient help and restraint the head of the patient can be manipulated into the desired position and no damage need occur to the apparatus.

In closed head injuries three routine views of the skull are necessary (1) anteroposterior, (2) right lateral and (3) left lateral A tangential view should be added when a depressed fracture is present or suspected

The plane which runs through the centre of each external auditory meatus and which cuts the lower margins of the orbit is the radiographic base from which the various accepted projections are made It is known as the orbitomeatal plane (Fig 94)

1 *Anteroposterior View.*—The patient is placed on his back and the head manipulated so that the orbitomeatal and sagittal

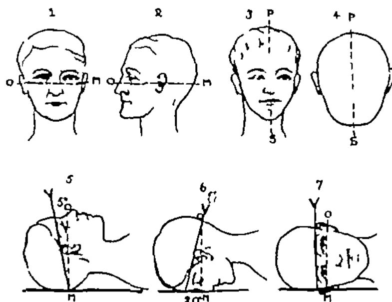


Fig 94

Radiological positions.

1 and 2, The orbitomeatal plane. 3 and 4 The parasagittal plane. 5, The anteroposterior view 6, The postero-anterior view 7 The right lateral view "X" indicates the axis of projection of the X rays.

planes of the skull are exactly perpendicular to the floor If the sagittal plane is allowed to rotate to one side an asymmetrical view of the skull will be obtained which is difficult to interpret. The rays are then projected so that they make an angle of 5° with the orbitomeatal plane (see Fig 94) The object of angulating the X ray tube is to bring the orbits low on to the films so that they do not obscure large areas of the vertex The frontal bone can be seen in greater detail by a postero-anterior than by an anteroposterior view, but this necessitates turning the patient on to his face a position he will rarely tolerate

2 *Lateral View*—In lateral views it has been agreed that the side of the skull nearest to the X ray film shall determine which is the right and which is the left lateral shoot *i.e* the right side down is the right lateral view Great care must be taken to obtain

true lateral view, otherwise the known landmarks of the skull become obscure. The sagittal plane must be parallel with, and the orbitomeatal plane perpendicular to, the ground (see Fig. 94). These conditions are most easily obtained with the patient on his face, but owing to restlessness the picture usually has to be taken with the patient supine, in which case the head must be forcibly turned to one side and the opposite shoulder raised on a pillow. When the patient vigorously refuses to have his head put into this position, the rays may be projected parallel to the floor with the patient's nose pointing upwards and with the film casette held against the opposite side of the head.

3. *Tangential View.*—In tangential views the head is so oriented that the suspected depression lies end-on to the projection of the X-rays.

Special views are necessary to demonstrate certain types of injury about the mastoid bones, petrous bones, paranasal air sinuses, and optic foramina, but as these examinations are rarely urgent, they may be deferred until the patient is well enough to be taken to the main X-ray department of the hospital. The radiological technique required for these various examinations has been described by Cairns and Jupe [1]

As a linear fracture may easily be confused with the natural markings in the skull, a differential diagnosis of the various radiological features will be found in the following table (Figs. 95-100) [2] A simple fracture line may remain visible for six months in children and for three years in adults.[3,4]

DIFFERENTIAL DIAGNOSIS OF VARIOUS RADIOLOGICAL FEATURES OF SKULL

Fracture Lines	Suture Lines	Meningeal Grooves	Diploic Channels
Clean-cut edges Run in all directions May cross suture and arterial lines Change *direction* abruptly Branch irregularly	Fine or dentate lines Run in constant positions May be widened by trauma or hydrocephalus	Fairly sharp margins Run in known directions Branch dichotomously Calibre diminished from below upwards	Fairly sharp margins Change course abruptly and form irregular patterns Often start in lakes near the sup long sinus Vary in width and often are beaded in appearance

[1] Shanks, S C, Kersley, P, and Twining, E W "A Textbook of X-ray Diagnosis" Lewis & Co London, 1938

[2] Wakeley, C P G, and Orley, A "A Textbook of Neuro-radiology" Baillière, Tindall & Cox London, 1938

[3] Stewart, W H "The Time Factor in the Disappearance of Roentgenographic Evidence of Fractures of the Skull" *Brit Jour Rad*, 1925, **30**, 399

[4] Vance, R G "The Healing of Linear Fractures of the Skull" *Amer Jour Roentgen*, 1936, **36**, 744



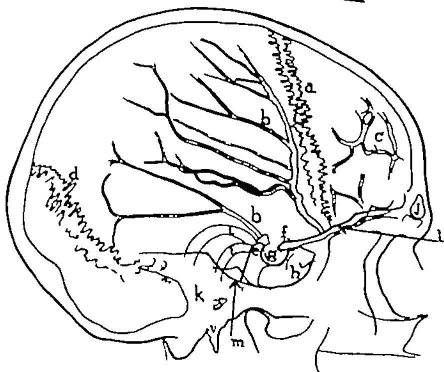


Fig. 93

The normal markings on the skull (lateral view).

a, Frontoparietal suture; *b*, meningeal grooves; *c*, diploic channels; *d*, occipitoparietal sutures; *e* posterior clinoid process; *f* anterior clinoid process; *g* pituitary fossa; *h* sphenoidal air sinus; *i*, shadows of the ears; *j*, frontal air sinus; *k* petromastoid bone; *l* floor of anterior fossa; *m* floor of middle fossa.



Fig 96.—Diploic channels may closely resemble fracture lines and are often mistaken for them This skull has not been fractured



Fig 97.—Stellate fracture lines Compare these with the meningeal grooves and diploic channels, both of which can be plainly seen



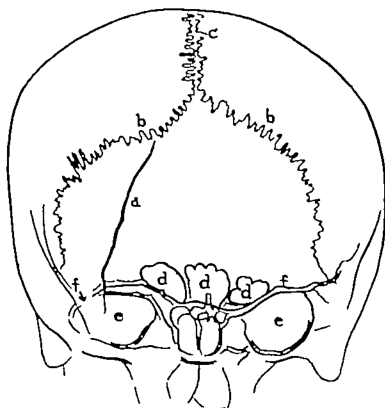


Fig. 98

The normal markings on the skull (anteroposterior view).
a Fracture line ; *b*, occipitoparietal sutures ; *c* intraparietal sutures ; *d* frontal air sinus ; *e* orbit ; *f* floor of anterior fossa.



FIG 99.—"Springing" of the occipitoparietal suture This is equivalent to fracture



FIG 100.—The fine linear type of fracture which is so commonly overlooked In a poor film it would have been overlooked.

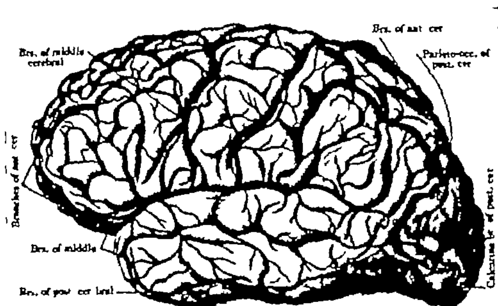


FIG. 101
Arteries of superolateral surface (*E B. Jamieson.*)

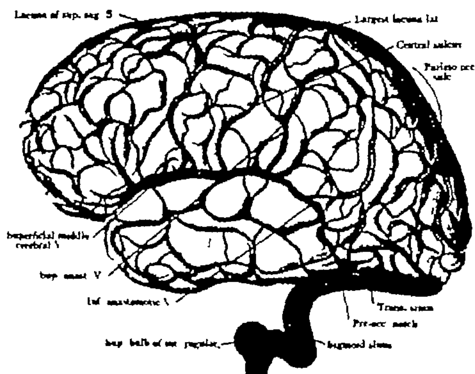


FIG. 102
Veins of superolateral surface and venous sinuses. (*E B Jamieson.*)

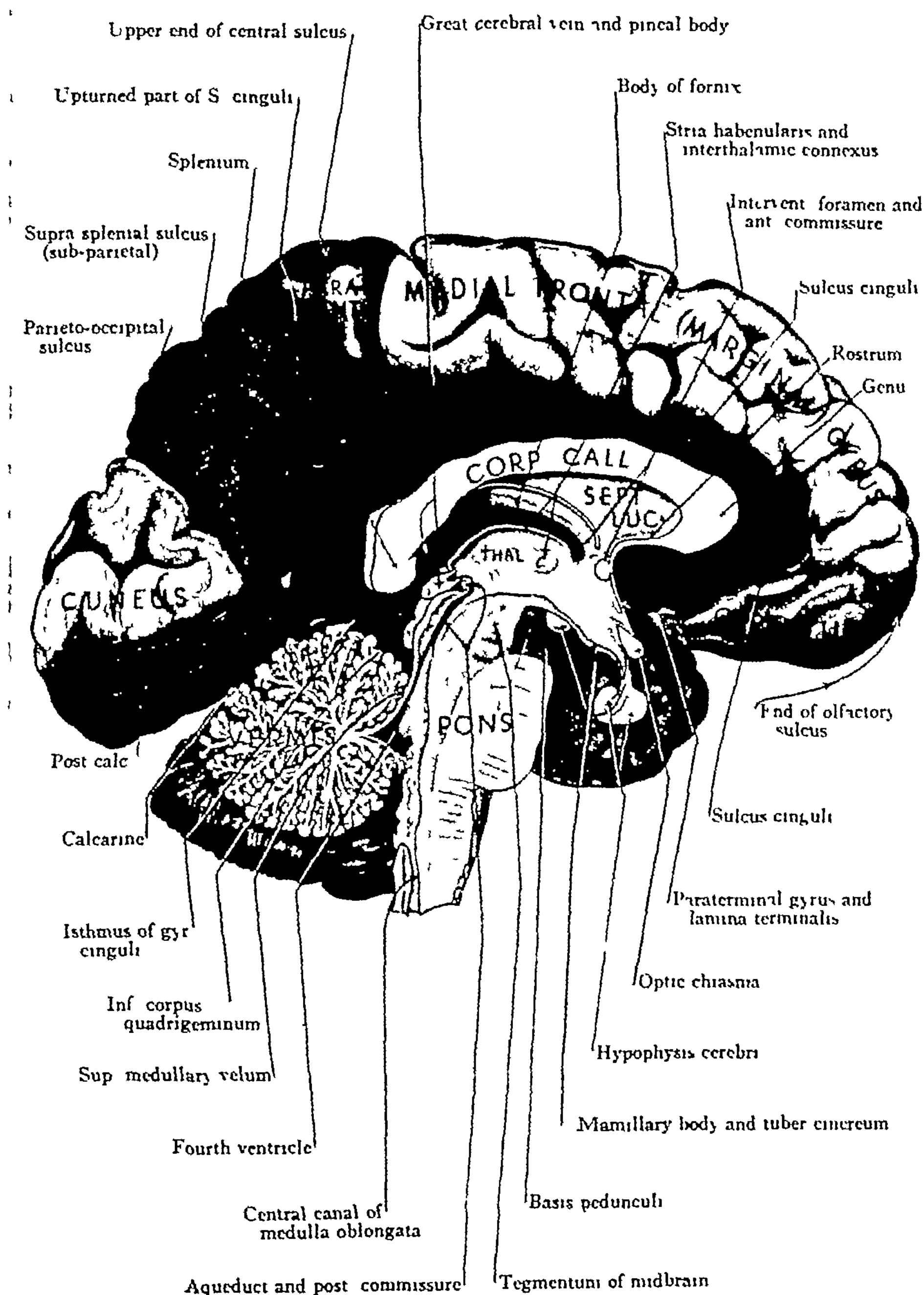


Fig 103

Median section of brain (*E B Jameson*)

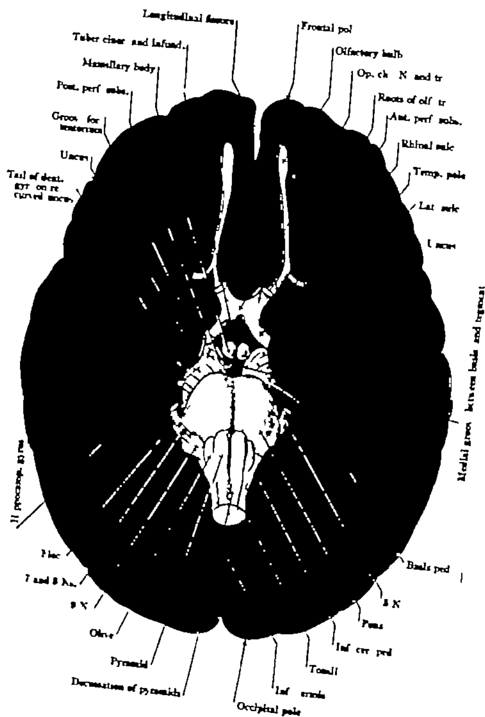


Fig. 104

Base of brain. (Brain hardened *ex situ* in order that the basis pedunculi might be seen.) (*E. B. Jamieson.*)

Signs referable to the Brain (Figs. 101-104).—Although the physiological functions of each part of the brain are so closely integrated that derangement in any one part makes its effect felt in all the others, certain nervous activities are dominantly represented in relatively circumscribed areas of the brain tissue (Figs. 105 and 106). If this were not true, anatomical localisation in diseased states would not be possible.

In non-traumatic neurological cases the pathological change is usually discreet enough to permit of precise anatomical localisation

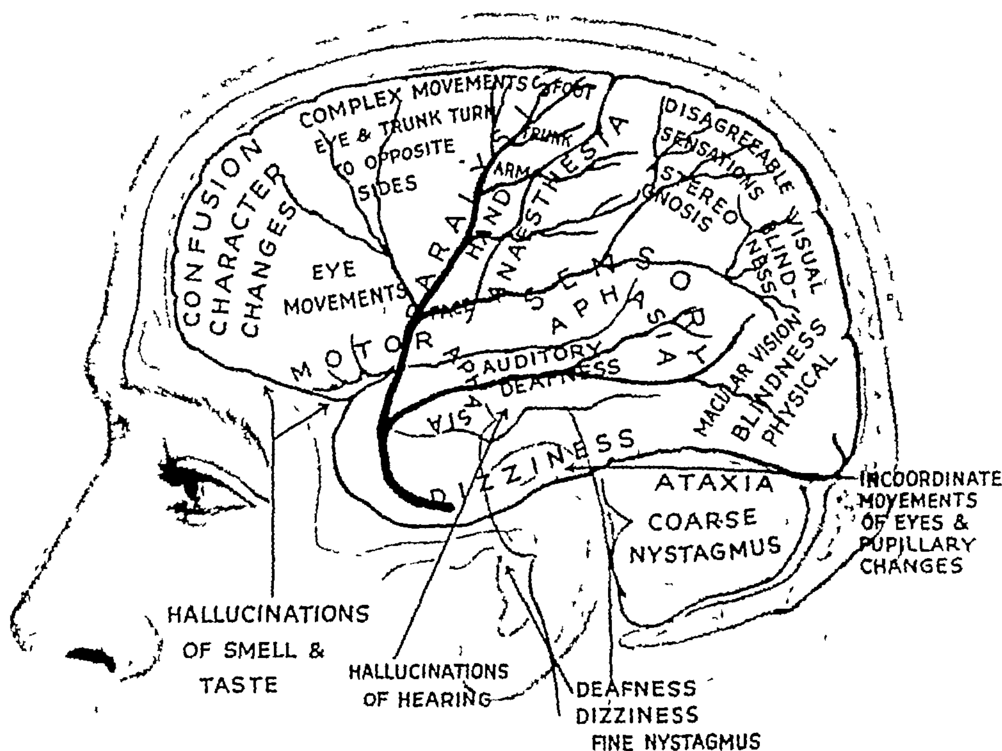


FIG 105

The localisation of function in the cortex of the brain

on clinical grounds alone. Diagnosis of the nature of the lesion, on the other hand, is rather more difficult, and is deduced from the mode of onset of the illness, from its progress and from the combination of symptoms and signs which are present at any particular phase in the life-history of the disease. Also, pathological diagnosis may be aided or confirmed by cytological, chemical and bacteriological examination of the cerebrospinal fluid, and by special investigations such as spinal manometry, encephalography or ventriculography.

In traumatic cerebral neurology, anatomical localisation and the diagnosis of the nature of the lesion is a much more difficult

problem than in non traumatic neurological cases, because the dominant features of the illness—unconsciousness and restlessness—have no precise localising value and no definite pathological significance. Moreover, there are no clinical syndromes which can with any certainty be attributed to contusion and laceration The onset of the cerebral illness is so rapid that it cannot be analysed save on those rare occasions when a latent interval occurs, and the illness may take an extremely variable course according to the development of secondary phenomena. Also, ventriculography and encephalography are, by the nature of the lesion, not so useful in traumatic as in non traumatic states. In many of the more serious cases their dangers preclude their use

It would therefore be wrong to attempt to oversimplify diagnosis in acute cerebral trauma as so many people have done in the past, for if it is not realised that the problem is essentially complex, bewilderment and not information will be the final result of most clinical examinations.

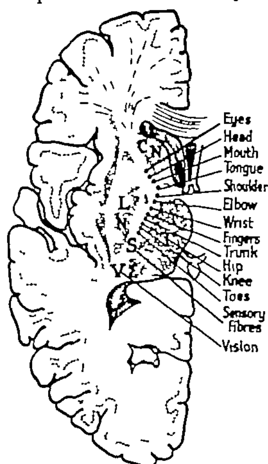


FIG 106

The disposition of the cortical projection fibres at the level of the internal capsule

CN Caudate nucleus; LN Lenticular nucleus; T Optic thalamus.

Confusion and Unconsciousness —The most important feature of any closed injury to the brain is unconsciousness It is a state of extreme gravity, and as long as it exists the patient's life is in danger When it comes on immediately after accident it is due either to diffuse neuronal injury or to damage to the ganglia of the brain stem, of the thalamus or of the hypothalamus, although it may be perpetuated by a secondary compression as the effects of the primary injury recede A period, however short, of consciousness immediately following the accident, as a rule means that any later ensuing state of unconsciousness is due to secondary developments, such as an increasing hæmorrhage which can be cured by surgical means. The slightest change in the depth of unconsciousness either one way or another is an infallible sign

of improvement or retrogression, and as recovery of consciousness is a gradual process, whether this be rapid or slow, repeated examinations at frequent intervals are necessary if changes are to be recognised early.

Depth of unconsciousness may be judged by the reactions of the patient to external stimuli and may be classified thus :—

Coma.—This is a state of complete unconsciousness in which there are no psychologically understandable responses either to external stimuli or to inner needs Certain primitive responses, such as the corneal, swallowing or tendon reflexes may or may not be present, and their absence is indicative of a very serious state within the lower centres of the brain. The patient cannot be roused or be compelled to make a movement by any kind of verbal command or by the infliction of pain, such as pressure on the testicle or of pricking of the finger-tips.

Semicoma.—In this state there is a complete lack of co-operation. The patient, however, can be made to make some kind of movement or to change his expression in response to painful or disagreeable stimuli. A test I have found most useful is to push the angle of the jaw forwards forcibly and to hold it in this position for a few moments Lack of response may be taken to mean coma When this manœuvre causes obvious discomfort, the patient is no more than semicomatose All the primitive reflexes, such as swallowing and closure of the eye on stimulation of the cornea, are present In coma there is retention of urine, possibly with overflow, whereas in semicoma the bladder empties reflexly whenever it becomes distended

Confusion.—By confusion is meant a clouding of consciousness. In this state the patient obviously makes an effort to think, but is unable to do so with any clarity or speed. There are, of course, degrees of confusion, and these may be graded according to the responses that can be obtained to commands In order to obtain some kind of uniformity in the assessment of confusion the Medical Research Council[1] have suggested that the following classification be adopted :—

Mild.—A state in which the patient, though presenting the characteristic feature of confusion in some degree, is capable of coherent conversation and appropriate behaviour.

Moderate.—A state in which the patient, though out of touch with his surroundings, can be got to give relevant answers to simple questions, such as "What work do you do?" "How old are you?" "Where do you live?"

Severe.—A state in which the patient, though for the most

[1] Medical Research Council "Glossary of Psychological Terms commonly used in Cases of Head Injury" M R C War Memorandum No 4 March 1941

part inaccessible, will occasionally show adequate response to simple commands forcibly given and, if necessary, reinforced by appropriate gestures, e.g., "Put out your tongue," "Take my hand"

The poles or association areas of the frontal lobes are thought to be concerned chiefly with the higher grades of the intellect and with the control of emotion, but they are not, as is so often believed, responsible for all kinds of thinking, and neither are they the parts of the brain in which consciousness is centred

So far, electrical or any other kind of physical or chemical stimulation of the cortex of the frontal poles has not yielded any useful information as regards the intellect, and what knowledge we have of the functions of the frontal association areas has been obtained from the effects either of pathological lesions in these areas or of lobectomies made in the operative removal of tumours Unilateral resections of that part of the brain which lies within the anterior fossa of the skull either on the left or right side do not lead to obvious intellectual changes as Penfield[1] and Jefferson[2] have shown apart from slight deterioration of initiative. Bilateral lobectomies, on the other hand, lead to a subnormal mentality, though according to Brickner,[3] who made exhaustive tests on one patient in whom both frontal poles had been removed, there are no specific functions in the frontal association areas which are not present to some degree elsewhere in the brain

In view of the above findings it is reasonable to suggest that confusion following cerebral trauma may occasionally be due to contusion of the frontal lobes and is a localising sign of injury to these regions

Let us consider the hypothetical case of a closed head injury where the brain stem only is damaged, the damage being of sufficient degree to produce coma When the brain stem fully recovers its function the patient will be fully conscious and the clinical neurological picture will be normal No doubt the recovery of the brain stem injury will be a gradual process, and, therefore, the clinical recovery will also be a gradual process one clinical picture supervening on another until full consciousness is attained It might be claimed that in many cases of head injury the dysfunctions of the brain stem centres are so dominant that the unfolding of the clinical picture is due solely to gradual recovery of the brain stem My opinion is that the evidence in

[1] Penfield, W. G., and Evans, J "The Frontal Lobe in Man: a Clinical Study of Maximum Removals." *Brain*, 1935, 58, 115.

[2] Jefferson, G "Removal of Right or Left Frontal Lobes in Man." *Brit Med Jour.*, 1937, 2, 199

[3] Brickner, R. M "The Intellectual Functions of the Frontal Lobes" 16, 354 The Macmillan Co. New York, 1936.

most cases is against this conception of the neural substratum of clinical recovery My belief is that the gradual recovery of consciousness and the unfolding of the neurological picture are best accounted for by the awakening of the many brain centres or levels from below upwards In broad clinical terms, coma is indicative of brain stem injury, semicoma of paralysis of the hemispheres and confusion of dysfunction of the frontal lobes of the brain. The variegated clinical pictures result from the variegated integrations of the possible level awakenings.

Posture and Movements.—There is little doubt that the functions of all the parts of the motor cortex are very closely associated with each other, and this leads us to a consideration of the integration of the nervous system as propounded by Hughlings Jackson, who is regarded as the father of modern neurology Basing his opinions largely on the activities of the motor system, Jackson came to the conclusion that the nervous system is built up in three different levels. The lowest concerns movements of the simplest form which are governed by the centres in the spinal cord and lower parts of the medulla. In Walshe's[1] words.

"The excitable motor cortex of the physiologist is the seat of the 'middle level' of the motor function. It has been evolved out of the lowest level and in it are represented, or reinco-ordinated, the simple and general movements represented in the lowest level. The re-representation consists in the synthesis of complex and special movements of the lowest level, and the experimental observations of Leyton and Sherrington have lent detail and confirmation to this conception. For Jackson, the 'præfrontal region' is the seat of his highest level. Here there takes place a further analysis and synthesis of movements represented in the middle level into the most complex and special movements of which the organism is capable.

"There are no 'abrupt' localisations in these two levels and *all* movements are widely represented throughout them"

If I may be allowed simpler terms, the lowest centres are where the pattern of each section of the movement machine is moulded and the excitable motor cortex where the whole machine is assembled. The præfrontal region represents the man who works the machine and makes it do the work for which it is intended.

As a higher centre is eliminated by injury, a lower functional level is released and becomes the integrating centre of the activities in the neurones below (Fig. 107) When the basal ganglia are eliminated there is poverty and slowness of movement, rhythmic tremor, rigidity, a fixed facial expression and diminished tendon reflexes Release of the system governed by the red nucleus leads

[1] Walshe, F M R "Syndrome of Premotor Cortex" *Brain*, 1935, 58, 75.

to contraction of those muscles which are concerned in maintaining the upright posture and to relaxation of the antagonists of these groups Postures determined by red nuclear influences are affected by proprioceptive impulses from the muscles of the neck and from the labyrinth These are known as the righting reflexes, and are so designed that the limbs and body are moved into the position of optimum balance according to movements and positions of the head Any lesion which cuts off the descending tracts from the red nucleus but leaves Deiter's nucleus intact leads to decerebrate rigidity In this state all the muscles of the body and limbs become rigid, so that the legs and arms act as props In man the hips and knees are fully extended and the ankles plantar flexed Sustained muscle spasm resists any attempt at passive movement and reflexes cannot be elicited The arms may be extended or flexed and they are held firmly in this position by agonist and antagonist muscle groups occasionally the body may be drawn into a position of opisthotonos Changes in position of the limbs, and particularly of the arms, may be brought about by changing the position of the head the requisite impulses in such cases coming from the labyrinths.

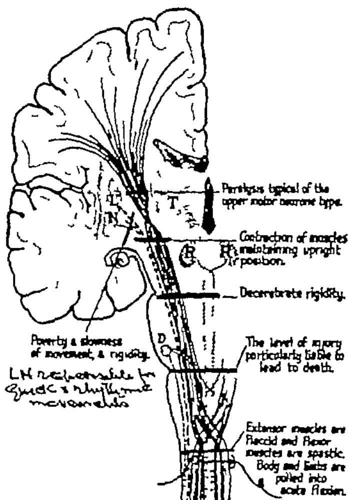


Fig 107

The pyramidal and extrapyramidal pathways. According to the level of interruption in these pathways different nervous phenomena ensue This is one of the main reasons why the clinical pictures in head injuries are so variable

L.N Lenticular nucleus; T Optic thalamus
R, Red nucleus; D, Deiter's nucleus.

When the centres below Deiter's nucleus take control the extensor muscles become flaccid and the flexor muscles go into spasm with the result that the limbs and body are pulled into acute flexion

The above neurological phenomena have been mentioned because they help to unravel the meaning of those peculiar postures and movements which are so often seen in the early phases of cerebral trauma and which cannot be explained by a superficial injury to the precentral area So often clinical observers unsuccessfully attempt to explain paralyses and postures purely by reference to the motor cortex where they know that the body is represented upside down with the hand to the mouth and with the leg over on the mesial side of the brain

After a severe head injury a patient may lie on his back with his jaw dropped and with his flaccid limbs taking up positions determined by gravity. Alternatively he may be curled up on his side and resent interference. Between these two postures any kind of position and type of movement may be seen Completely flaccid limbs and a fallen jaw usually mean that the whole nervous system is in a state of severe shock and if improvement does not rapidly take place the patient will die. A patient who has been only slightly dazed will look as though he is asleep in the ordinary way His position will be comfortable and the tone in his facial muscles will be good Prognosis is usually favourable when a patient lies curled up on his side and resents being moved from this position.

Decerebrate rigidity is a most important sign When it occurs immediately after an injury it means that the upper part of the brain stem has been contused, but when it develops after an interval it is due to a tentorial herniation, the result of some secondary development such as œdema or a massive hæmorrhage. This state may suggest the onset of meningitis, particularly if the head is drawn backwards or if the temperature rises, and both these signs may be present in decerebrate rigidity. A lumbar puncture should never be done to establish a diagnosis, as removal of cerebrospinal fluid from the spinal theca will lead to further impaction of a pressure cone. Differential diagnosis is usually not difficult, since in decerebration the patient lies still and the muscle rigidity is persistent, whereas in the early stages of meningitis the patient is often restless and muscle tone variable.

Except in coma, movements of all four limbs are usually spontaneous or can be produced by suitable stimuli. Muscle tone is often changeable, so that at one moment a joint can be manipulated easily through a full range of movement, whereas at the next the muscles may be in a state of spasm Reflexes are also variable The knee jerks may or may not be active, and the plantar response may fluctuate from flexor to extensor at short intervals These observations point to damage of the motor pathways, but unfortunately they are of no precise localising

value and are best explained by rapid changes of the circulation within the areas concerned

A paralysed limb lies motionless in spite of suitable stimuli. In the early stages of paralysis due to any cause, the muscles are flaccid and the limb falls heavily to the bed with a slap when raised and allowed to drop without support. Comparison with the fall of a better controlled non paralysed limb will demonstrate this flaccidity even more closely

Convulsive seizures or epileptic twitchings in any group of muscles localise the injury to the opposite Rolandic cortex They

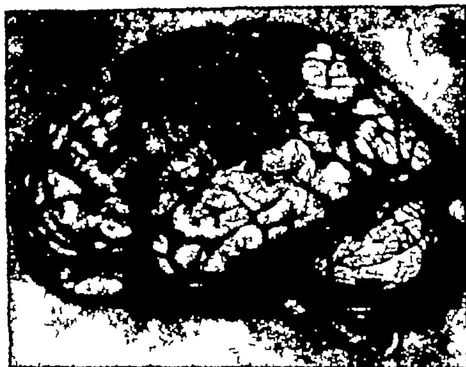


Fig. 108

A rare example of a high convexity laceration resulting from a closed injury A persistent hemiplegia resulted in this case

may indicate a contusion or irritation by a subdural or extradural clot, although on clinical grounds it is impossible to differentiate between the two

Hemiplegias are much more common than monoplegias, and they may occur immediately or develop after an interval A hemiplegia immediately following an injury is best regarded as due to bruising of the motor cortex, especially in children Even if the diagnosis proves to be incorrect, no valuable time will have been wasted as regards recovery of power if an operation has to be performed later Very occasionally severe laceration of the motor cortex occurs in closed injuries and leads to permanent paralysis (Fig 108) Interval hemiplegias may be caused by

(1) subdural or extradural hæmorrhages, (2) œdema, (3) external hydrocephalus and (4) exhaustion following a convulsive seizure. These possibilities must be eliminated, if necessary by direct inspection through a trephine hole before a diagnosis of thrombosis of the middle cerebral artery is made. Thrombosis of the middle cerebral artery occasionally occurs and leads to extensive cortical atrophy, which can be demonstrated by ventriculography or encephalography. A paralysis due to a tear of the brachial plexus is an occasional happening and is difficult to diagnose, particularly as paralyses, both of peripheral and central origin, are flaccid in the early stages. A differential diagnosis, however, is possible. In the peripheral type the skin is insensitive and there is no reaction when it is pinched or pricked with a pin, whereas in paralysis of central origin the skin is sensitive, and when a painful stimulus is applied to it, even though the limb itself cannot be moved, some kind of response occurs elsewhere in the body—possibly a change of expression or a sweeping movement of the other arm. Later, when spasticity or wasting has occurred, the diagnosis is unequivocal. Also, peripheral injuries are often accompanied by evidence of bruising or thickening in the triangles of the neck

Restlessness is one of the most common features of any injury to the brain In many cases a patient lurches from one side of the bed to the other as if seeking a more comfortable position, or he pushes and pulls at the bedclothes incessantly. Often he attempts to get out of bed in a meaningless kind of way. What the significance of all this activity may be is difficult to know. Probably it is the reaction of the patient to the meningeal pain of a subarachnoid hæmorrhage, associated with bruising of the frontal lobes.

In delirium, movements are incessant, excessive, writhing in character and apparently purposeless. Whether this is the result of pain, hallucinations or delusions is not known, but it is always a grave sign and indicative of severe bruising of the brain, associated with profuse subarachnoid bleeding.

Normal posture and smooth, purposeful co-ordination of movements are indicative of the milder types of cerebral injury and usually mean that the chances of recovery are good.

Aphasia.—Aphasia is an inability, not due to unconsciousness, either to use or to comprehend language in any of its forms

There are two varieties—sensory or receptive, and motor or expressive—and in many cases there is a combination of both these conditions in which one or other is dominant.

Sensory aphasia was localised by Wernicke to the region of the left supramarginal gyrus in right-handed people, and it is characterised by an inability to understand the spoken or written word.

Motor aphasia is loss of power to speak, write or draw, although the messages of writing and speech can be understood. Motor aphasia was localised by Broca to the region of the posterior end of the left inferior convolution of the frontal lobe but there is much evidence to show that a more extensive area of the brain is concerned

Many types of aphasia exist, and formal terms are given to certain types in which a particular modality of language is effected Dysphasia implies that the command of language has been impaired but not entirely lost. Agraphia means inability to write alexia inability to read, and apraxia difficulty in knowing how to use a simple machine or object, such as scissors or a pencil

All degrees and types of aphasia are encountered in acute cerebral trauma, and recognition of this condition is important not only because of its localising value but because it may give the illusion that unconsciousness is deeper than it really is A warning must be given that if an important decision has to be made as regards surgical exploration on the sign of aphasia, it is imperative to seek from some third person the information as to whether the patient is right handed It is unwise to assume that he is right handed, because in one of my cases had this precaution not been taken, the wrong side of the skull would have been opened and a large subdural hæmorrhage overlooked

Motor aphasia is frequently mistaken for confusion, although the differentiation of the two states is not particularly difficult. An aphasic patient has a conscious look about him, and when spoken to some kind of intelligent expression comes into his face, and at best he will obey a complicated request. A confused patient on the other hand, looks bewildered and can understand only a simple command He ignores anything difficult or complicated

When a patient comes to a stage of consciousness sufficient to permit recognition of aphasia, he has a very good chance of surviving his injury and most probably will eventually recover his ability to speak with little residual impairment. In two cases only have I known aphasia of any noticeable degree persist, and these were due to extensive depressed fractures which had not been raised by surgical means.

Differentiation between sensory aphasia and severer grades of confusion is impossible In the phase of cerebral irritation a patient may talk incoherently when stimulated and give the impression to his relatives or to unskilled observers that he has gone out of his mind It is very gratifying to be able to say at an early stage with confidence that this state will probably subside and that the patient will not be mentally impaired Occasionally,

on the sign of aphasia, I have been able to localise a progressive lesion and to centre a surgical exploration correctly for the removal of a surface clot.

Aphasia is a most reliable localising sign but its pathological cause is often a matter of conjecture. It may be due (1) to contusion or laceration of the areas concerned, (2) to compression by a clot or depressed fragment of bone, (3) to œdema or (4) to thrombosis of a large vessel, and a diagnosis has often to be made through an inspection hole.

Position and Movements of the Eyes.—In concussion a patient's eyes are usually closed. When they are not it is a sign either of approaching consciousness or coma, and a differentiation of these two states is a very simple matter. In coma the corneal reflexes are absent or sluggish, whereas in the nearly conscious patient they are extremely active. Other tests for determination of depths of unconsciousness are unequivocal.

In those cases when it is necessary to open the eyelids passively the patient will often resist and try, by some kind of purposive movement, to escape the irritation of light or to avoid the unpleasantness of having his eyelids manipulated. The more purposively the patient resists, the nearer he is to consciousness. For example, a sweep of the hand is indicative of a more complex intellectual process than a mere movement of the head away from the painful stimulus. In spite of resistance a patient's eyelids should be kept open long enough to permit observation of the position of the eyes and of their movements. When movements of the eyes do not occur spontaneously, they may be induced by moving the patient's head gently from side to side or by twisting his body.

Abnormal positions of the eye and inco-ordinated or restricted movements may be produced by one of the following conditions :—

(i) Hæmorrhages into the orbit.
(ii) Damage to the extra-ocular muscles or destruction of their pulleys by fractures of the orbit.
(iii) Contusion, severance or avulsion of the third, fourth or sixth cranial nerves.
(iv) Injury to the ocular nuclei or their connecting pathways.
(v) Labyrinthine and cerebellar concussion.
(vi) Impairment of function in the visuo-psychic fields.

Displacement of the axis of the eye is due either to extensive extra-orbital hæmorrhages or to deformity of the orbital cavity by fracture of its walls (Fig. 109). Bony damage to the orbit is a common occurrence and is due to the frequency with which patients are thrown on their face. Usually the orbital floor is

broken, so that the eye sinks downwards Upward displacements are rare, but some degree of lateral displacement is often seen

Squints may be complete or incomplete, fixed or changeable. A complete and fixed strabismus is due to damage of an extra-



Fig 109

A fracture of the lateral wall of the orbit may not only lead to displacement of the eye but to injury of the external rectus muscle or its nerve supply with resulting diplopia.

ocular muscle or to contusion laceration or avulsion of the nerve which supplies it (Fig 110) An extra-ocular nerve may be damaged in any part of its course, although this usually occurs close to the brain stem *or within the orbit*

Skew deviations are often seen and are due to both eyes being

pulled away from the axis in which they would normally be resting at any given moment. In other words, a skew deviation is a bilateral strabismus in the vertical plane. Such ocular divergencies are usually associated with inco-ordinate wanderings of the eyes in which both globes wobble spontaneously about the orbit without any reference to each other. These conditions of abnormal posture and movement are due to changes within the brain stem affecting the ocular nuclei or their connecting pathways. Hæmorrhages into the brain stem in concussion are known to be common, but these rarely destroy the ocular nuclei, because very few patients are left with a permanent strabismus even after a severe head injury. Circulatory disturbances within the brain stem probably account for most skew deviations and inco-ordinations of movement. Also, a vascular basis explains why they are so rapidly changeable in form and degree and why they so often disappear without leaving any residual disability. Occasionally the eyes are fixed in the middle axis and nothing that may be done in the way of stimulation will induce them to move. In these cases the injury involves the posterior longitudinal bundle which is the main co-ordinating tract for the ocular nuclei.



Fig 110
A third-nerve paralysis due to a fracture involving the sphenoidal fissure

Fine nystagmus or rapid and regular oscillations of the eye are of frequent occurrence and are due to concussion of the labyrinth. Coarse nystagmus is rather less common and is due to contusion of the cerebellum. In this case the oscillatory movements of the eyes have a wider range than in labyrinthine nystagmus, and there is a slow swinging component away from the point of fixation followed by a rapid corrective jerk backwards

When in the state of confusion a patient will often look at the examiner in a meaningless kind of way, staring vacantly forwards. Also, his sight cannot be directed towards an object or activity which would attract the gaze of a normal person. Occasionally this state is due to apraxia and results from damage to the visuo-psychic centres.

Of all the neurological signs which occur in acute cerebral trauma abnormal postures and movements of the eyes are the

most frequent, apart from unconsciousness and restlessness, and indicate that the brain stem is usually affected when a patient is concussed

The Pupils—The pupils may be contracted or dilated Often they are unequal, and rapid alterations in their size are common. Usually they react to light by constriction, but occasionally may dilate in a paradoxical way on stimulation. Such pupillary changes are indicative of an injury to the brain stem and have much the same meaning as alterations in the movements of the eyes. In other words, they point to intrinsic rather than to



Fig. 111
In a fixed dilated pupil the consensual, as well as the direct, light reflex is absent.

compressional changes, and although of pathological importance they are of no great surgical significance

The so-called "Fixed Dilated Pupil of Hutchinson," however, is a most important sign, of both localising value and pathological inference In this condition the pupil is fully dilated and it does not contract when a bright light is shone into it or into the opposite eye (Fig 111) It is necessary to prove that the consensual as well as the direct, light reflex is absent before diagnosing Hutchinson's pupil, because in an eye blinded by an intra ocular hæmorrhage or by severance of its optic nerve the pupil is dilated and does not contract to light although it does so when the opposite retina is stimulated A fixed dilated pupil" is an infallible sign of raised intracranial pressure, and means that a tentorial pressure cone has developed which is compressing the brain stem and stretching the oculo-motor nerve at the point where it is about to enter the wall of the cavernous

sinus Surgically its significance is of paramount importance, for if the uncinate herniation is not relieved actively the patient is almost certain to succumb, and, as will be shown later, explorations on both sides of the skull may be necessary in these cases

Small fixed pupils which remain contracted in spite of changes in the intracranial condition often indicate a hæmorrhage into the pons, and such a diagnosis is more than probable when bilateral pyramidal signs are present and when the temperature rises suddenly and continues to rise

Pulse and Blood Pressure.—In the early stages of primary shock the pulse is rapid and thready and the blood pressure is low, but often when the patient has been put to bed and warmth applied the circulation rapidly improves. When stasis of capillary circulation persists in spite of accepted methods of resuscitation, the prognosis is very grave, and the patient will probably succumb within twelve or, at the most, twenty-four hours. Raised intracranial tension causing medullary compression may produce a compensatory rise in systolic pressure without improvement of the diastolic pressure, which remains dangerously low. Depth of shock may thus be masked if diastolic pressure is not known.

In states of mild confusion the pulse rate and blood pressure are usually within normal limits, and, given that the mental condition is improving, they are of no particular prognostic or diagnostic value Furthermore, in those conditions where the patient becomes gradually more drowsy the circulation does not in the early stages deteriorate, as compensations within very wide limits are possible. Therefore, in the presence of other signs of deterioration of the intracranial conditions, a favourable pulse rate and pressure may be misleading.

In states of severe confusion and in semicoma the pulse rate is usually raised to between 90 and 110 beats per minute, whereas the blood pressure, as far as can be judged without comparison with a pre-accident reading, remains within normal limits [1] Gradual changes in depths of unconsciousness in coma or in semicoma are very difficult to appreciate, and often the first indication is an alteration in pulse pressure and pulse rate. A patient may remain unconscious for several days without any apparent variation in his mental condition, and so long as his circulation does not deteriorate there is every reason to hope that he will recover Alternatively, as soon as the pulse rate begins to rise and its volume to fail the whole picture changes and the chances of recovery are poor

[1] Woodhall, B "Acute Cerebral Injuries Analysis of Temperature, Pulse and Respiration Curves" *Arch Surg*, 1936, **33**, 560

A fast bounding pulse is a sign of raised intracranial pressure It is often seen in comatosed patients with stertorous breathing, and indicates embarrassment of medullary circulation. It is, of course, a much more favourable sign than a weak thready pulse, as it means that vigorous efforts of compensation are being made to maintain the essential cerebral circulations

Bradycardia or a slow pulse is a condition far more often described than seen, and is in fact a very great rarity in the acute phases of a head injury In convalescence it occasionally occurs, but is of no particular diagnostic or therapeutic significance On the other hand, it is of very great importance when a patient is unconscious, as it means that the brain is being severely compressed and probably by an extradural or subdural hæmorrhage As a working rule a combination of a slow pulse, below sixty beats per minute, and unconsciousness must always be taken to mean an extradural hæmorrhage until proved otherwise

In middle meningeal hæmorrhages in which there has been a latent interval the pulse rate and blood pressure usually show the following changes While the patient is conscious they are normal, then, as drowsiness develops, the pulse rate increases and blood pressure rises. As unconsciousness deepens, the pressure rises and the pulse rate falls and may become as low as forty beats a minute Occasionally the pulse rate remains high and never falls below normal Finally, as the cerebral circulation becomes inadequate, the systemic circulation also fails, with the result that the blood pressure falls and the pulse becomes rapid and thready.

(7) **Temperature** —When first brought in to the wards from the streets, patients are shocked and chilled and consequently their temperatures are subnormal Later, as heat is applied, body temperature rises, and in coma a patient may very easily be overheated if the temperature of the bed and room in which he is being nursed is not carefully regulated

In semicoma, apart from environmental influences, a rise of temperature of 1° or 2° F is common and is due to absorption of extravasated blood Fluctuation of temperature within these limits in the first few days is usual and is of no particular surgical significance A secondary rise after the temperature has been stabilised for a day or more is often a very serious sign, as it may indicate renewed subarachnoid bleeding or the development of pneumonia or meningitis

After severe intrinsic injuries of the brain patients often die in hyperthermia. The temperature rises as soon as shock has passed, and it continues to do so in spite of cold sponging and

may reach as high as 111° F. before the patient succumbs. Any rise of temperature above 101° F., whatever the depth of unconsciousness, is a very grave sign, as it is so often indicative of a severe intrinsic injury to the brain or of a profuse subarachnoid hæmorrhage.

Differences of one or two degrees of temperature between the two sides of the body are occasionally found and are due to interference with the sympathetic nervous system, but they throw no light on the exact site or on the nature of the injury.

Papillœdema.—Papillœdema is so rarely seen in the early phases of concussion that routine retinoscopy is apt to be omitted. This omission is a serious mistake, since swelling of the optic discs, when it does occur, is an unequivocal sign of raised intra-cranial pressure.

When papillœdema develops in the acute phases of cerebral trauma, immediate relief of pressure by intravenous dehydration, spinal drainage, exploration or decompression is indicated, particularly when there are signs of deepening unconsciousness or other evidence of retrogression.

Absence of papillœdema does not mean that intracranial tension is not raised, as drainage of the retina is not impaired until pressures higher than those usually found in cerebral trauma are reached. In acute expanding lesions, such as middle meningeal hæmorrhages, retinal evidence of pressure no doubt would be more often seen if the patient did not succumb rapidly without surgical intervention.

In my own experience papillœdema has never occurred before the third day, and only in cases of obstructive hydrocephalus and generalised œdema.

Respiration.—Normal respiration is a good prognostic sign, as it means that the brain has not received a severe intrinsic injury and is not being seriously compressed by a surface hæmorrhage.

Increased rate and depth of breathing which occur in restlessness can be explained by physiological adjustments rather than by pathological processes.

Stertor is usually a sign of impending death. It is seen in the early stages of coma when loss of muscle tone allows the jaw and tongue to fall backwards to impede respiration and occurs just before respiration is about to fail.

Deviation from normal rhythm indicates failure of medullary circulation, and the patient will almost certainly die if the nature of the lesion is such that it cannot be relieved surgically.

The autonomic or respiratory centre proper is a complex

structure and is thought to be situated in the pons and upper part of the medulla. Extremely different types of breathing develop when transections of the brain stem are made from above downwards. The upper part of the centre is concerned with normal or pneumotaxic breathing, the middle with apneustic and the lower with gasping breathing. Apneustic breathing occurs when the transection is made just below the inferior colliculi and the rhythm is as follows. First the patient takes a slow, deep inspiration which he holds for one or two minutes. This is followed by a sudden relaxation of the inspiratory muscles and the air is suddenly expressed from the chest. After a few normal breaths the apneustic cycle is repeated.

In gasping breathing, inspiration and expiration begin and end suddenly and are followed by a pause. This type of breathing occurs when the transection is made at the level of the striæ acousticæ. A transection at the calamus scriptorius causes cessation of respiration.

Breathing in middle meningeal hæmorrhages and other expanding lesions first becomes deep and fast, then deep and slow, and finally irregular. Changes in irregularity throw light on the functions of the complicated respiratory centre, and it is often educational to sit and listen to alterations in breathing in a comatose patient as the various centres fail from above downwards.

Vomiting.—Vomiting is a common occurrence in head injuries. Unfortunately it has no definite neurological or pathological significance. In the early phases following accident it is thought to be due to irritation of the gastric mucosa by swallowed blood. In the later stages of illness it is regarded as being central in origin. The manner in which a patient vomits is important of course. If, when he is desirous of vomiting, he sits up in a purposive way and uses a receiver or turns his head over the side of the bed, this means that he is not deeply unconscious. On the other hand, when a patient lies perfectly still on his back and fluid stomach contents well up into and trickle over the side of the mouth, this is a sign usually of deep unconsciousness and of grave prognosis.

Intracranial Pressure.—Apart from direct inspection of the brain through surgical exposures, spinal and ventricular manometry are the only reliable methods of measuring intracranial tension, since deductions from symptoms and clinical signs are apt to be misleading.

Measurements are made in millimetres of cerebrospinal fluid, normal pressure in the lateral position registering between 50 mm. and 150 mm.

INTRACRANIAL PRESSURE IN TWO HUNDRED CASES OF TRAUMATIC UNCONSCIOUSNESS

Pressure of C S F	Number of Cases
Over 300 mm	20
Between 200 to 300 mm	146
Normal	30
Decrease	4

All these readings were taken within twenty-four hours of the injury

In the above series the highest pressures were found in restless patients whose cerebrospinal fluid was heavily stained with blood On one occasion over 500 mm was registered, and in this case the blood-stained cerebrospinal fluid shot out of the top of the manometer as if the spinal needle had been introduced into the abdominal aorta Low normal or subnormal pressures occur particularly in shock . in old people with poorly nourished bodies ; in the later stages of coma ; and in those cases where there has been a loss of cerebrospinal fluid from the nose, ears or into the subgaleal space.

Usually intracranial pressure becomes stabilised within normal limits after one or two spinal drainages in which sufficient fluid is withdrawn to bring the pressure to 50 mm.

From spinal manometry alone no precise deduction can be made regarding the pathological state within the cranium, since increased pressure may be due to (1) extravasated blood, (2) increased volume of blood within the cranial vessels, (3) cerebral œdema, (4) hydrocephalus

Furthermore, depth of unconsciousness cannot be judged by spinal manometry alone [1]

Russell [2] found that on twelve occasions when the pressure was above 200 mm. the patients were fully conscious, whereas in seven stuporous patients the pressure was below 200 mm Also, in large extradural hæmorrhages subnormal tensions may occur, as Jefferson has shown In my experience a low or subnormal intracranial tension in comatose patients is a bad prognostic sign and probably indicates that the cerebral circulation is failing. Alternatively, when intracranial pressure is high a stuporous

[1] Paterson, J H "Some Observations on the Cerebrospinal Fluid in Closed Head Injuries" *Jour Neurol*, 1943 July and October, vol vi

[2] Russell, W R "Discussion on Intracranial Pressure its Clinical and Pathological Importance" *Proc Roy Soc Med*, 1934b, **27**, 832

patient's life is still in the balance, and efforts to save it will often be rewarded by gratifying results

Changes of pressure in a conscious patient are of very little significance as regards life and death because the underlying cause, whatever this may be, is so often amenable to treatment.

Although intracranial pressures above 500 mm are often found in cases of tumours of the brain in which consciousness is not impaired, this does not mean unconsciousness cannot be produced by the smaller rises of tension in cerebral trauma. It is the speed at which the pressure develops rather than its magnitude which is important Rapidly expanding lesions do not give time for compensation of cerebral circulation to take place as is the case in those which develop more slowly

In one case of an acute subdural hæmorrhage which was unassociated with intrinsic injury of the brain, I was able by spinal manometry to demonstrate a gradual rise in intracranial pressure up to 300 mm, at which point the patient died (Decompression was not done in this case because the patient succumbed within half an hour of admission to hospital whilst preparations for operation were being made.)

If, as is generally believed, increased intracranial pressure, as distinct from the pressure of a large clot, has a deleterious effect on recovery of local or diffuse contusions of the brain, treatment ought to be designed to keep the intracranial pressure within normal limits Whether this is done by decompression, lumbar and ventricular drainage or by intravenous dehydration, depends on the cause of the rise in pressure Intravenous dehydration, for example, can only be of value in cases of cerebral œdema, in other cases it is harmful

The presence of blood in the cerebrospinal fluid of course, establishes the diagnosis of subarachnoid hæmorrhage and, as stated previously, will be found in 75 per cent. of cases of severe concussion It does not, however, localise the lesion or distinguish between venous and arterial bleeding The amount of blood present is of prognostic value only within wide limits. According to Russell, patients are always comatose when more than 100,000 red blood cells per cubic centimetre are found in the cerebrospinal fluid

In my own experience profuse bleeding associated with unconsciousness has usually ended fatally, and the greatest quantities have been found in gunshot injuries In one case in which a bullet passed through the whole head the blood clotted in the test tube as it was withdrawn through the spinal needle Raised intracranial pressure in the absence of subarachnoid bleeding is indicative of œdema or of hydrocephalus.

5 c.c. and replaced by injections of similar amounts of air It is preferable to tap both lateral ventricles, and in this case 5 to 10 c.c. of air on each side is all that is necessary to give a satisfactory picture. By means of ventriculography, *enlargement, collapse,* deformity or displacement of any part of the ventricular system can be clearly visualised (Fig 113)

Encephalography —Encephalography is the replacement of cere-



Fig 113

A ventriculogram showing displacement of the left ventricle to the left side. In this case it was not possible to tap the right ventricle presumably because of displacement.

brospinal fluid by oxygen or air through a spinal puncture performed either at the level of the cisterna magna (cerebello-medullary) or at the lumbar theca In this investigation the patient is put on the operating table and a lumbar puncture performed in the left lateral position Cisternal punctures are better avoided in restless patients lest an uncontrolled movement cause the needle to prick the medulla. A block or suitable support is then fixed behind the patient's thigh or buttocks and the table tilted at least 45°, with the head uppermost Ten cubic centimetres of fluid are then withdrawn and replaced by an equal amount of whichever gas is chosen for injection At least 45 c.c of air must be introduced and more if detailed pictures are considered necessary The advantage

of oxygen over air is that it is more rapidly absorbed and post-operative discomfort is less severe. Long experience has shown that air taken from the room and not subjected to any special processes of sterilisation may be injected without causing meningitis.

The radiographic advantage of encephalography and ventriculography is that the gyri and cisterns, as well as the ventricles, are outlined.

One of the great advantages in diagnosis of tumours of the brain is that ventriculography can be used freely with reasonable safety, and is always permissible when localisation is in doubt.

On the other hand, in acute cerebral trauma its use is restricted by its attendant dangers and is indicated only on special occasions.

The main indications for ventriculography are .—

(i) When unconsciousness has lasted a whole day at the same depth and is then followed by retrogression.
(ii) When depth of unconsciousness has not changed after thirty-six hours.
(iii) Failure to expose a suspected subdural hæmatoma through inspection holes bored in the skull.

The main indications for encephalography are .—

(i) Lack of localising signs when a large clot or any other form of compression is suspected.
(ii) When definite localising signs, such as hemiplegia or aphasia, are present

Ventriculography is a safer procedure than encephalography for the following reasons :—

(i) Less air or oxygen needs to be injected to give useful pictures
(ii) The gas can be removed easily at the end of the examination
(iii) There is less likelihood of a pressure cone being precipitated.

Properly used, ventriculography is a means of saving life, and at the moment there is a growing opinion in authoritative circles that ventriculography should be used more frequently than has hitherto been customary, particularly since drainage of the cerebrospinal fluid through a ventricular tap is the best method of maintaining intracranial pressure within normal limits.

Electro-encephalography.—When a man's eyes are closed and his mind is at rest, oscillations of electrical potential occur on the

surface of the head These consist of a series of waves with a frequency of ten per second and an amplitude of 0·05 to 0·1 millivolts

Berger[1] first described these oscillations in 1929 and they are now known as the Berger Rhythm According to Adrian and Matthews,[2] they arise in the occipital cortex and are neutralised when other parts of the brain become active Waves of other shapes and frequencies have been described, and in 1936 Grey Walter,[3] working at the Maida Vale Hospital for Nervous Diseases, put these discoveries to direct use, as a result of which the electro-encephalogram is becoming a routine part of neurological diagnosis Pathological states alter the electrical activities of the part of the brain concerned and, after suitable amplification such alteration can be measured and located by correct placing of electrodes

The apparatus is not simple, and a great deal of experience is necessary to interpret the records An important advantage of this method over air encephalography however, is that the patient is put to little inconvenience and no harm is likely to be done. All that is necessary from his point of view is that his scalp and hair are cleaned

A cerebral tumour is electrically dead compared with normal cortex and when on the surface of the brain, can be located with some certainty In head injuries, however, the problem becomes rather more difficult because the injury is diffuse In my own opinion the primary use of the electro-encephalogram is in the detection of large surface clots. Later, should complications occur, it is of value in localising an epileptogenic focus and in throwing light on the nature of the neuronal discharge.[4-7]

INDICATIONS FOR SURGICAL TREATMENT

Even after the most careful examinations it is not always possible to interpret the many clinical signs observed in terms of pathological lesions with that precise accuracy necessary to guide successful treatment It is therefore proposed to review the diagnostic problem from a different angle even at the risk of

[1] Berger H. *Arch. f. Psych.*, 1929 87 527

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[3] Grey Walter W "Electro-encephalography in Cases of Cerebral Tumour" *Proc. Roy Soc. Med.*, 1936-37 30, 579.

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Short A. Rendle and Dunster M *Brit Med Jour.*, 1940, 1, 884.

[6] Williams, D "Electro-encephalogram in Acute Head Injuries. *Jour Neur and Psych.*, 1941 4, 107

[7] Williams, D Electro-encephalography in Chronic Post-traumatic States. *Jour Neur and Psych.*, 1941 4, 131

repetition. From the clinical standpoint all cerebral injuries may be classified into three main groups according to the degree of unconsciousness when the patient is first seen, and according to the way in which unconsciousness developed.

Group I.—Group I, which is always the largest in any clinical series, includes those patients who are conscious or who are rapidly approaching consciousness when first seen.

Although opportunities to observe the earliest phases of concussion rarely occur, it is important to be acquainted with the sequence of events in this stage, as it elucidates many of the problems which may arise later and gives a guide to the correct form of treatment

In a typical case a man receives an injury to the head and either falls or remains on the ground completely unconscious. In this stage there is complete paralysis of function which, as Symonds has stated, may extend for a moment to the vital centres Then after a few moments recovery begins in order of sequence from the lowest to the highest neurological levels. First, the pulse and respiration return, but the patient remains in a state of coma with flaccid muscles. Possibly this is the phase of medullary control. Soon the muscles regain their tone and the reflexes return, which probably means that the upper part of the brain stem is recovering its function. Then, as the cerebrum recovers, consciousness returns. The patient moves a limb, then makes a purposive movement, usually with his hands, and soon tries to sit up. At this stage he becomes restless, attempts to speak, but is confused and often becomes resistive and even violent. Before final recovery he reaches the stage of automatism, which is a condition of extreme medico-legal importance, for although he behaves naturally and is apparently in full control of his faculties, he is not really responsible for his actions He can stand up, can converse logically and answer questions correctly. The danger is that, unknowingly, he may make statements which are untrue He may start up his car and drive carelessly or he may behave dangerously in some other way to himself or others without later being aware of what has happened Statements taken immediately after concussion should be accepted with a good deal of circumspection In the less severe cases a patient may be dazed for a moment only Things go black before his eyes, but before he falls or loses his balance he regains his senses and goes on as usual

The state of slight concussion in games is very well known and many of us have experienced it. A player receives a knock on the head and is dazed for a few moments but does not leave the field. He continues to play, but automatically and badly. He misjudges the ball in a way he would not do in the ordinary course

of events, and does not show that initiative or sense of anticipation which has gained him his position in the team.

When a conscious patient is admitted to hospital giving a history of concussion, no particular medical or surgical treatment is necessary if there are no residual signs of injury to the nervous system, and the only decision to be made is whether he should be detained or not.

This decision is important, because a patient who has apparently made a complete recovery from concussion may suddenly develop a fatal secondary compression. It is safer, therefore, that all patients who have received an injury to the head of sufficient degree to bring them to hospital should be kept under observation for a few hours, and those who have been definitely unconscious should be detained for at least one night. If this is done it can be claimed that reasonable care has been taken whatever may happen later in the way of complications. An X ray of the skull is also advisable because of the importance the public attaches to a fracture. Finally, on discharge from hospital, a message should be sent to the patient's general practitioner requesting him to send the patient back to hospital if severe headache or drowsiness develops.

When local brain damage has occurred a patient should be admitted to hospital at once for full neurological investigation. Signs and symptoms of local injury, as Symonds has pointed out, are associated most frequently with those of generalised brain injury, but they may occur with little or no disturbance of consciousness. Also, they vary in kind and number according to the site and nature of the injury. Cranial nerves may be avulsed, special centres may be contused and important blood vessels ruptured or thrombosed.

The results of injury to special parts of the skull and brain will be discussed in a succeeding chapter, but for the sake of continuity the subject will have to be introduced here.

Blows on the top of the head may cause paralysis of both legs. Recently I have seen three such cases, and on each occasion the patient was badly dazed by the blow but soon regained consciousness and was co-operative at the time he reached hospital. Both legs were spastic, the knee jerks were increased and the plantar responses were extensor. Sensory changes were present, although these were subjective rather than objective. On testing, no area of sensation was definitely lost, the feeling being that a lighter stimulus was being applied than to the normal parts of the body. In other words, the skin felt as though it were being touched through the clothes. Gordon Holmes[1] described this

[1] Holmes, Gordon, and Sargent, P. "Injuries of the Superior Longitudinal Sinus." *Brit. Med. Jour.*, 1915, 2, 493.

syndrome in detail during the last war and suggested that some of these cases are due to occlusion of the superior longitudinal sinus, which interferes with drainage of the motor cortices, thus explaining the bilateral signs.

Hemiplegia and monoplegia may also follow an injury which causes no more than momentary loss of consciousness and, as in the case of unconsciousness, may occur at the moment of violence or develop after an interval.

Immediate paralysis may be caused by one or a combination of the following conditions: (1) neural shock, (2) exhaustion following epileptic seizures, (3) compression by depressed bony fragments, (4) contusion and (5) laceration. Bony compression may readily be confirmed or eliminated by radiography. In the other cases diagnosis depends largely on the subsequent events. Recovery from neural shock and epileptic exhaustion is usually rapid and often complete within twenty-four hours. Paralysis due to contusion usually begins to improve within a few days and full recovery of function is the rule. Prognosis is serious in lacerations. Fortunately this is a very rare condition in non-fatal closed injuries and is invariably associated with a closed depressed fracture.

Interval paralysis may be due to (1) compression by a surface hæmorrhage, (2) compression by an external hydrocephalus, (3) œdema, (4) arterial occlusion and (5) spreading venous thrombosis. Prognosis as regards recovery of motor power is very much worse in delayed than in immediate paralysis, because intrinsic lesions are much more common than surface compressions. The onset of paralysis is often progressive, save in the case of intracerebral hæmorrhage which is ushered in as an acute cerebral crisis Delayed post-traumatic intracerebral hæmorrhages often occur in middle-aged people with normal blood pressures. Typically, the episode occurs in the patient's bedroom. He feels giddy, attempts to get out of bed and falls. When he is lifted it is found that he is paralysed. Full consciousness soon returns. Spinal pressure is not appreciably raised, and neither blood nor abnormal chemical constituents are found in the cerebrospinal fluid Encephalography, however, shows a filling defect in the lateral ventricle on the affected side. Considerable recovery may be expected in these cases, but a certain amount of clumsy movement and impairment of the finer movements of the fingers will almost certainly remain In secondary paralysis, diagnosis of the underlying cause should always be made by inspection through trephine holes or preferably by air replacement

Group II.—Group II consists of those patients who are unconscious when first seen, but who have been conscious during

some period after the accident. This is a very small group, but it is important from the surgical point of view. Whether the patient was unconscious before the latent interval is of prognostic importance, since absence of early unconsciousness means absence of intrinsic brain damage.

Meningeal Hæmorrhages—The latent interval in middle meningeal hæmorrhages, as shown in the previous chapter, is usually a number of hours only, although it may be as long as a week or more. The first symptom is increasing headache, and this must never be treated lightly when a history of an injury, however slight, has been given. Giddiness, mental confusion or drowsiness must be regarded as pathognomonic and not merely as suspicious signs of extradural compression. As the hæmorrhage increases, drowsiness or confusion changes to unconsciousness and signs of pyramidal impairment develop on the opposite side of the body. At first the limbs become weak and spastic. Convulsive twitchings may occur at any stage either early or late. The reflexes are typical of those of an upper motor neurone lesion, the knee jerk is increased, the abdominal reflexes on the same side are diminished or absent, and there is an extensor plantar response. Respiration, at first fast and deep, slows and then becomes irregular. The pulse in a fully developed case is slow, *i.e.*, below sixty beats a minute, and the blood pressure is high. A fixed dilated pupil may or may not be present and in any case is usually a late sign.

When pyramidal signs are confined to one side of the body, lateralisation of the hæmorrhage is easy, but if the clot is not evacuated at this stage bilateral motor signs rapidly appear and diagnosis is much more difficult. Often a patient is first seen when bilateral signs are well developed. In these cases there may be (1) bilateral spasticity, (2) spasticity on one side and flaccidity on the other or (3) bilateral flaccidity. These possible combinations are explained by the way the clot compresses the brain. First the cortex on the side of the hæmorrhage is irritated, and thus may lead to convulsive seizures in the contralateral limbs. Then venous congestion develops at the site of compression and causes a spastic type of paralysis. Later the cortex becomes ischæmic and a spastic changes to a flaccid paralysis. Finally, as the clot expands, a similar sequel of phenomena occurs on the opposite side of the brain and the combination of clinical signs found at any time depends on the phase of compression.

In a combination of flaccid and spastic paralysis the clot, therefore, is on the side of the brain opposite to the limbs showing signs of flaccid paralysis. In cases in which there is no difference between the two sides of the body, lateralisation of the clot on

physical signs alone is impossible, but the diagnosis may be made if the sequence of events is known.

Subdural Hæmorrhages.—In subdural hæmorrhages the latent interval is usually longer than in middle meningeal hæmorrhages, and signs of compression may not develop for days or even years. Repeated or increasingly severe headaches associated with mental changes, such as drowsiness or apathy, are the first indications of compression Later signs and symptoms are extremely variable. Periods of unconsciousness may come and go, papillœdema may or may not be present and localising signs are often absent The important fact in the diagnosis of a chronic subdural hæmatoma is that a relatively slow-developing cerebral crisis has occurred at some time after a complete or a partial recovery from an injury. In those cases when it is not possible to localise a clot on neurological evidence, such as a hemiplegia or a facial paralysis, it must be found by inspection through exploratory trephine holes. Usually it is discovered high over the parietal lobes or just above the Sylvian points.

When a large blood clot cannot be found in spite of exploration, encephalography or ventriculography is indicated, but if the facilities for this procedure are not ideal it is best to perform a right-sided subtemporal decompression and then to seek expert aid.

Group III.—Group III consists of those patients who are unconscious and have not been conscious at any time since the accident

Cases of Favourable Prognosis.—In this sub-group the patients are not deeply unconscious. At most they are confused or drowsy, and although they may resent interference or be unable to co-operate in a complicated examination, they will obey a simple command or imitate a simple gesture. Restlessness may be present, but this is not incessant or of a lurching character, and often the patient will lie in a perfectly natural position for long periods. Occasionally he will attempt to get out of bed in a purposeless kind of way or in an attempt to empty his bladder. All the primitive reflexes, such as swallowing, are present and, apart from the mental changes, there are no obvious abnormal neurological signs Pulse, respiration and blood pressure are normal

Patients suffering from minor neuronal injury or minor contusion are conscious and out of danger usually within twenty-four hours of the receipt of their injury.

Cases of Grave Prognosis.—Many of the patients who are obviously going to die are moribund from the beginning and make no kind of improvement despite all the usual methods of resuscitation Unconsciousness is so deep that no kind of response

can be elicited even by very painful stimuli The corneal reflexes and tendon jerks are absent, the muscles are flaccid, the jaw is fallen and, in the worst cases, swallowing is absent. The pulse is fast and feeble, but may for a short period be bounding before it begins to fail Respiration may be stertorous at the start, but soon becomes irregular and later feeble Often the skin is wet with perspiration Within an hour or so the temperature begins to rise, signs of basal pneumonia develop and the patient dies in hyperthermia within twelve or twenty four hours

Cases of Doubtful Prognosis—This is the most important group from the diagnostic and therapeutic points of view and the one in which detailed and repeated examinations must be made if logical conclusions are to be drawn The patients are either in coma or semicoma and there are neurological signs referable to damage to the motor system, but there is nothing definite to point to a focal or compressive lesion. In the first twelve hours, therefore, in these cases it is usually necessary only to treat shock and to apply surgical first principles, as will be described later At the end of this period some cases will obviously be improving, others will be moribund and there will be some in which no apparent recovery or retrogression has taken place. It is this latter group which engages chief attention. From the twelve-hour period onwards a knowledge of the intracranial pressure, as measured by spinal manometry, is necessary, since it is believed that the optimum conditions for recovery of damaged cerebral tissue are produced by maintenance of the intracranial pressure within normal limits Also, repeated examinations are essential so that the development of localising neurological signs or changes in depth of consciousness are observed at the earliest possible moment.

Let us start at the stage when there is mental and physical paralysis. One of the earliest signs of recovery is return of normal tone in the flaccid muscles, and this can be most easily observed by a return of facial expression Later there may be a spontaneous movement of a limb or there may be an active change from one position to another Reactions to painful stimuli appear as the patient recovers and the first groan is the first sign of recovery of function of the speech centre. With further improvement, restlessness develops and the patient incessantly changes his position Whether this is the result of physical pain or of a state of subconscious mental anxiety is not known, but probably both factors play an important part

Irritability follows restlessness and may be expressed either by word or deed The patient will often violently resist by purposive movements any kind of handling however gentle and will

knock away a cup or spoon as an attempt is made to feed him. Later he confines his objections to the spoken word. He is rude, impatient, intolerant and lacks all sense of deference, as if there had been a complete break-up of his personality At times his behaviour may be indecent.

Then comes the stage of mental confusion. The patient begins to waken and wonder what has happened. Although he is able to co-operate to some extent, he is unable to engage in sustained rational conversation At one moment he will recognise his relatives and make some intelligible statement, whereas at the next he will show no interest in them and begin to mutter incoherently or irrelevantly. He may sometimes lose emotional control and cry or laugh with equal facility The most common feature is that he does not realise the gravity or the meaning of his condition and is apt to pick at a wound or try and break down a splint controlling a fracture Insight for a time is in abeyance and confabulation is common. Finally he comes within the limits of intellectual normality, and although mentally sluggish he realises he has received an injury and can assess its importance for both himself and his family His facial expression changes from one of indifference to one of concern and he begins to complain of his aches and pains.

In favourable cases respiration, temperature and the circulation remain within fairly normal limits; any divergence from the normal, even though the mental processes are clearing, is serious, as it may indicate meningitis or pneumonia

If at any time after twelve hours the patient becomes more deeply unconscious and the circulation fails, a decision has to be made as to whether or not this deterioration is due to the development of secondary phenomena which can be treated by cranial exploration or by decompression of the brain.

This decision is difficult, and it would be misleading to give the impression that subtemporal decompression plays a large part in treatment, it does not It plays a small though important part, and its correct practice marks the difference between those who are and those who are not experienced in treating acute injuries to the brain

Neuro-surgeons are not agreed on the question of surgical exploration in closed head injuries; while some discountenance it, others advocate frequent explorations and decompressions through large osteoplastic flaps. The following suggestions are based on my personal experience

INDICATIONS FOR SUBTEMPORAL DECOMPRESSION

1 **Retrogression, following a Period of Improvement, which cannot be controlled by Spinal Drainage or by Intravenous Dehydration.**—In this type of case the general condition of the patient progressively improves in a way that cannot be explained by recovery from shock Unconsciousness becomes less deep, the muscles regain their tone, primitive reflexes become firmly established and abnormal reflex responses disappear Pulse, respiration and blood pressure remain within, or approaching, normal limits. Usually before these observations can be established a period of at least twelve hours is necessary Then, for some reason which is not very obvious, the patient s condition begins to deteriorate and, in particular, the depth of unconsciousness increases Reliable localising signs are rarely found

When there are no localising or lateralising signs, a right sided decompression should be made. Also, two inspection holes should be sunk on the left side—one at the Sylvian point and one over the parietal eminence in order to eliminate a possible extradural or subdural hæmorrhage. Such conditions, when present on the right side, will be uncovered by the decompression On those occasions when a large surface hæmorrhage is found, it is not enough merely to remove the clot without decompressing the brain, as the absence of a latent interval means that the brain has received intrinsic damage and probably will become œdematous if it is not already in this state

2. **Delayed Decerebrate Rigidity** —When a tentorial herniation develops within twelve hours it usually means that the brain has received a severe intrinsic injury, and very little benefit is to be gained by subtemporal decompression. On the other hand, when decerebrate rigidity develops after twenty four hours, the causative herniation can often be relieved successfully by surgical decompression A fixed dilated pupil may be present to lateralise the hernia, but when this is absent a bilateral exposure has to be made At the time of the operation, swelling of the brain usually prevents anything more than a simple opening of the dura, but if the temporal lobe can be lifted without laceration or bruising of its cortex the hernia may be lifted out of the hiatus tentorii or the tentorium may be split from within outwards.

It is in this type of case that the most spectacular results can be obtained

3 **A Fixed Dilated Pupil.**—A fixed dilated pupil even in the absence of decerebrate rigidity, indicates a raised intracranial pressure of such degree that it will lead to a fatal outcome if not

relieved. If after twelve hours—and it is impossible to judge in a shorter period than this—the patient is not improving and his state is serious, then a decompression ought to be made on the side of the affected pupil.

4. **Prolonged Unconsciousness associated with Persistently High Cerebrospinal Fluid Pressure.**—After thirty-six hours, if a patient has shown no sign of recovery of consciousness and the cerebrospinal fluid returns to a supranormal figure, in spite of repeated spinal drainage or of intravenous dehydration, it is advisable to perform a right-sided subtemporal decompression.

INSPECTION HOLES

In the last few years I have been making frequent use of small inspection holes cut in the skull to establish a diagnosis in doubtful or difficult cases. These holes are placed at points determined by neurological signs, and are made with a trephine so that the disc of bone may be replaced if nothing abnormal from the surgical point of view is found.

McConnell of Dublin has also used this method extensively, and on many occasions he has found a subdural collection of fluid the drainage of which has led to obvious and rapid relief of symptoms.

His routine in unconscious patients is to place a hole 1½ to 2 in above the external auditory meatus so that it can be incorporated in a subtemporal decompression if this is found necessary. In those cases where surface hæmorrhage is suspected but cannot be established on clinical data, an inspection hole will save many hours of unnecessary anxiety from the surgeon's point of view, and particularly as it is a procedure that can be carried out rapidly without doing any harm to the patient even if a negative exploration is the result.

Inspection holes are the only means by which a definite diagnosis can be made of massive hæmorrhage not associated with a latent interval. In all cases where diagnosis is in doubt, limited explorations under local anæsthesia are indicated.

FURTHER CLINICAL CONSIDERATIONS

Even though the diagnosis of closed cerebral trauma has been discussed both from the view of assessment of individual neurological signs and of clinical generalisations, the problem is so difficult and important that consideration from a third aspect is justifiable. It is proposed, therefore, to describe individual

or small groups of cases illustrating in particular those points which lead to correct therapeutic procedure. Some repetition is unavoidable, but even this might be useful

MIDDLE MENINGEAL OR EXTRADURAL HÆMORRHAGES

(*a*) **A Typical Case** —At two o'clock in the afternoon of 26th May 1942 a boy aged twelve years was riding his bicycle when he lost his balance and fell to the ground According to witnesses, he struck the right side of his head heavily against the road surface He lay helpless on the ground and had to be carried to the pavement, where he remained unconscious for about five minutes Soon, although dazed, he was able to stand on his feet, and later recovered sufficiently to remount his bicycle and ride home. His complaints at this time were of a vague discomfort in the head and a feeling of drowsiness. On his own initiative he went up stairs, undressed and got into bed Later, when his mother came home she found him sleeping, and on wakening him found he was weak on the left side of the body She took him at once to her doctor who fully examined the boy and sent him home to rest in bed, instructing the mother to seek further medical advice if he did not progress. The next day the child was difficult to waken, but his mother thought he was merely in a healthy sleep On 28th May 1942 the doctor found him to be definitely unconscious, and on his advice the boy was transferred to the University Hospital, where he was admitted at three o'clock in the afternoon The Resident Medical Officer diagnosed a right sided middle meningeal hæmorrhage, and at 5 10 P M on the same day the boy was transferred to the neurosurgical unit.

On admission he was drowsy, but could be roused to some extent by loud and repeated commands He was persuaded, for instance, to attempt to raise his arms The whole of the left side of the body was weak, the arm and leg falling heavily to the bed on being lifted and then released A similar test was carried out on the right side, and the limbs fell more slowly and were obviously under some degree of control There was increased muscle tone on both sides, but it was more marked on the right than on the left. Both knee jerks were sluggish the abdominal reflexes were weak on both sides, the left plantar reflex was frankly extensor, the right being doubtful The left side of the face was weak, the left side of the mouth being drooped in repose and the left facial muscles reacting less vigorously than those on the right side as each cornea was stimulated On the right side there was a complete third nerve paralysis (Fig 114) The right eyelid was drooped the eyeball was pulled into the outer canthus

and the pupil was dilated and fixed. On the right side of the head there was a boggy swelling in the region of the temporal fossa. The pulse rate was 56, the blood pressure 120/80, temperature was subnormal; respiration was 16 per minute.

A diagnosis was made of a right-sided extradural hæmorrhage, probably due to rupture of the middle meningeal vessels.

At operation the temporal muscle was swollen and stained with blood, there was a large diagonal fracture of the temporal bone. There was an extradural hæmorrhage due to rupture of the middle meningeal vessel 1 in. above the point where the vessel turns upwards from the base on to the vertex The blood clot was centred over the anterior half of the temporal lobe and was about 1 in. thick It extended into the anterior fossa, downwards

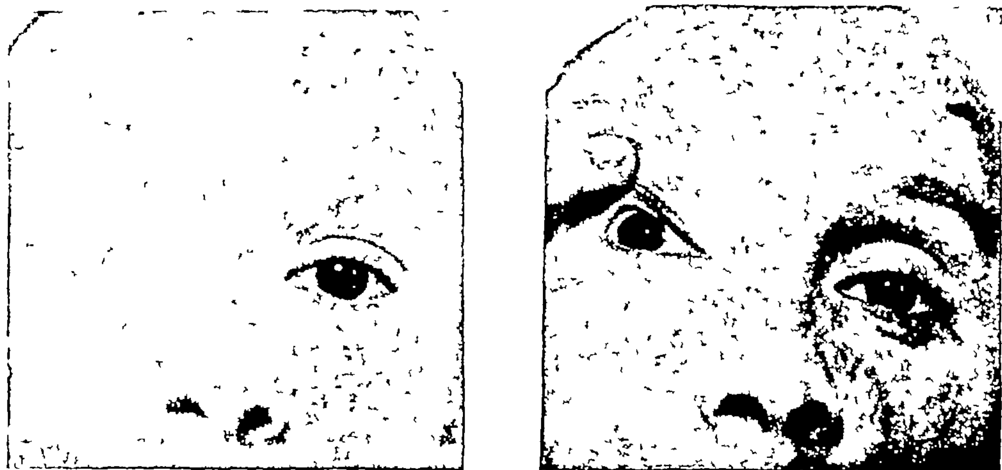


FIG 114
A third-nerve paralysis due to a tentorial herniation.

on to the floor of the middle fossa, and far backwards and upwards on to the parietal lobe.

Comment.—In this case the state of unconsciousness immediately after the fall was almost certainly due to concussion; that is to diffuse neuronal injury. From this condition the boy made a rapid recovery The latent interval, as is usual, was not symptomless From the moment of recovery from the first attack of unconsciousness the boy was aware of a fullness in the head and that he was not thinking as clearly as usual, conditions which prompted him to go to bed.

The gradual increase in his symptoms was sufficient to point strongly to the correct diagnosis which can often be made before consciousness is impaired. Headaches of rapidly increasing severity and the onset of giddiness strongly suggest bleeding. The slightest degree of confusion or stupor always confirms the diagnosis

beyond doubt. It is very dangerous to allow a patient to become deeply unconscious before coming to a conclusion, as this seriously jeopardises the success of operative treatment. Once a diagnosis has been made, no delay must occur before the compression is relieved To prevent confusion when the final clinical picture of the above case was reviewed, two diagnoses—the pathological and the anatomical—had to be kept clearly in mind

Pathological Diagnosis—The diagnosis of an extradural clot was made entirely on the history of an undoubted lucid interval Signs of third nerve involvement—fixed dilated pupil, muscle paralysis and dropped eyelid—were merely confirmatory evidence of compression. In cases in which there is doubt about the diagnosis, the presence of a fracture line crossing the middle meningeal groove is presumptive evidence of an extradural hæmorrhage

Anatomical Diagnosis—If an operative exposure is to be centred correctly, then the site of the clot has to be localised with precision In the case under consideration the weakness on the left side of the body and the third nerve paralysis on the right side pointed clearly to the clot being on the right side of the head That it was low and due to bleeding from the middle meningeal vessels was suggested by fullness in the right temporal fossa, since this sign is indicative of an underlying fracture of the bone When in doubt, and it was not in this case, the presence and site of a fracture can be established by radiography It must never be assumed that when an extradural clot is diagnosed and lateralised that it is necessarily situated in the classical position about the pterion

Post-operative progress in this case was gradual, it being two days before the boy was fully conscious Recovery of power in the left arm and leg was apparently complete long before the reflexes reverted to normal Regarding the third nerve paralysis, the eyelid began to open first, then the squint lessened, and finally the pupil contracted and its reaction to light returned

(*b*) **With Associated Local Brain Damage.**—On 24th November 1942 a man walked into the accident room of a hospital stating that he had just been knocked off his bicycle by a motor vehicle He was dazed and there was an abrasion of the scalp in the right parietal region When seen by a medical officer he was able to co-operate even to the extent of giving his name and address It was thought advisable to admit him By the time he reached the ward he was deeply unconscious and could not be roused His breathing was stertorous, and all his limbs were stiff in the position of extension It was thought the man was going to die.

When I first saw him an hour or two later his condition had somewhat improved He reacted to pricking of the skin, and when spoken to loudly would mutter a few incoherent words. He could not co-operate in the simplest neurological examination —for example, he would not try to raise his limbs from the bed. The left limbs were weaker than the right, as judged by the way they fell to the bed when raised and allowed to fall. The pupils were equal, of mid-dilatation, and reacted slowly to light There were inco-ordinate movements of the eyes when the lids were opened. The ankle and knee jerks were active and equal on both sides, as were the arm jerks The abdominal reflexes were absent on both sides The plantar reflexes were doubtful on both sides. There was a boggy swelling on the right side of the head in the temporal region. The pulse rate was 80, the blood pressure 130/80, respiration was 18 per minute and regular in rhythm

At 7 45 P.M. the same day the man was admitted to the neuro-surgical unit, transport over a distance of a few miles having done him no harm The neurological picture had changed very little; the man still was in a condition of semicoma X-rays revealed a transverse fracture in the right temporal region crossing the middle meningeal groove.

At operation at 8 P.M the same day a right subtemporal exploration was made via a subperiosteal muscle slide. The findings were as follows :—

(*a*) Hæmatoma in the right temporal muscle.
(*b*) Transverse fracture of squama of temporal bone.
(*c*) Large extradural hæmorrhage due to rupture of the middle meningeal vessels on the rim of the base of the skull.
(*d*) Torn dura mater
(*e*) Subdural hæmatoma
(*f*) Small laceration of outer surface of temporal lobe and subarachnoid clot.

Immediately following removal of the blood clots and repair of the wound the man's condition was no worse. He was still in semicoma, but could be made to move his right leg and arm freely The left side was densely paralysed. The pulse rate, after the bandage had been applied and the man left undisturbed for fifteen minutes, was 80. The blood pressure was 120/80, and respiration was 30 per minute.

The following day he was less deeply unconscious, but the weakness on the left side was still obvious. It was not until 12th December 1942 that he became fully conscious. During the intervening period he had been incontinent of urine. The weak-

ness on the left side slowly recovered over several weeks Finally a complete recovery was made both mentally and physically, and the man returned to his work as a painter

Comment—The sudden loss of consciousness, followed by spontaneous and rapid improvement, was very suggestive of an epileptic seizure, and thus was the diagnosis originally made However, the man did not recover full consciousness as was to be expected if epilepsy had been the only factor operating Therefore, in view of the lucid interval a surface hæmorrhage was suspected, the diagnosis being confirmed by evidence of fracture of the temporal bone. The pulse rate, it is important to note, was never below 80 pre-operatively, which means that bradycardia is not an essential sign of cerebral compression Epilepsy, it must be remembered, often results from sudden cerebral compression, although in this case it might have been caused by the laceration in the temporal lobe Sealing of the ruptured middle meningeal vessels and removal of the extradural clot alone in this case would not have saved the man's life No doubt in cases of middle meningeal hæmorrhage lives are lost because concomitant lesions amenable to surgical procedure are overlooked

A case of a man aged thirty four years, from whom there was removed a posteriorly placed extradural clot of sufficient size presumably to account for symptoms of recurrent drowsiness, further illustrates this point. As a routine, the dura was carefully inspected and was found to be discoloured a bluish green On incision a subdural clot was evacuated which was greater in size than the extradural hæmorrhage Possibly, therefore, it would be wise to open the dura in every case of extradural hæmorrhage in order to inspect the subdural space

(*c*) **With Associated Extensive Brain Damage Case 1**—On the evening of 3rd August 1943 a man aged twenty two years received an injury to his head during a brawl when he fell to the ground following a blow on the jaw Apparently he was concussed and, according to witnesses, he never again recovered complete consciousness before being transferred to hospital the following day at 1.30 P M He was admitted to the neurosurgical unit at 4.20 P M On examination he was found to be in a state of severe confusion bordering on semicoma, but apparently was no worse than he had been some hours earlier He was restless and resented examination He appreciated pin prick and would mutter unintelligible answers to questions shouted in his ear He moved all his limbs freely, but it was judged that his right side, including the face, was weak. The arm, knee and ankle jerks were brisk and equal Both plantar reflexes were extensor The abdominal reflexes were brisk on the left side and sluggish on the right The

pupils were equal in size and of mid-dilatation. Both reacted briskly to light. The eyes did not wander aimlessly. When they did move, the movements were co-ordinated; they were expressionless, another sign of impaired consciousness. Respiration was normal in rate and rhythm; blood pressure was 130/70; the pulse rate was 70 and the temperature 98°. There was a large bruise at the back of the head. X-rays of the skull showed springing of the left parieto-occipital suture.

It was impossible, from the clinical picture, to be certain on one examination whether or not an extradural clot was present From the history it was judged that unconsciousness had remained at a fixed level for more than twelve hours. This is always suggestive of cerebral compression, because diffuse neuronal injuries after twelve hours usually show signs either of deterioration or of improvement. It was decided to observe this man for a further period before making a final diagnosis.

Three hours later his condition had slightly deteriorated; he was more difficult to rouse and became more unruly when being examined. The pulse rate was 68; respiration was 20, the blood pressure 120/80 and the temperature was subnormal. Neurologically there were no definite localising or lateralising signs. On the evidence of deepening unconsciousness, following what might be regarded as a relative lucid interval, a diagnosis was made of cerebral compression due to an extradural hæmorrhage. The clot was presumed to be in the left occipital region on the evidence of the posterior bruising of the scalp and on the radiographic sign of springing of the left occipitoparietal suture.

At operation a large extradural clot was found, covering the left occipital lobe and extending into the posterior fossa Bleeding was found to be emanating from the bone and from those small venous channels which run from the bone into the lateral sinus. The blood clot was removed and bleeding points controlled, either by plugging wax into the open diploic channels or by placing muscle grafts over the ruptured sinus veins.

As far as one could judge, the cerebral compression had been relieved, but in spite of this the man's condition gradually became worse. There was increasing stupor, the pulse rate rose and the man died ten hours later in hyperthermia

A post-mortem examination showed—

(*a*) The extradural clot had not reformed.
(*b*) Extensive bilateral subdural hæmorrhage.
(*c*) Subarachnoid hæmorrhage.
(*d*) Small lacerations on the under surface of both frontal lobes.

(e) Contusion of the tip of the right temporal lobe

(f) Contusion of the under surface of the left cerebellar lobe.

Comment.—The above example illustrates that group of important cases in which there is no lucid interval but in which a steady level of unconsciousness is maintained for a considerable period followed by gradual retrogression. Exploration in this type of case is always indicated even though there may be severe and concomitant brain damage, because in some instances the relief of compression will be sufficient to sway the balance successfully in favour of the patient and save his life.

Case 2.—The history is that of a middle-aged man having received an injury to his head while at work, as a result of falling to the ground from a scaffolding. His friends went to his aid immediately and found him unconscious. Within half an hour of the injury he was transferred to hospital.

On examination he was found to be comatose, breathing was shallow and rapid, the pulse was rapid and thin and the temperature subnormal. He was put to bed, treated for shock and examined in detail an hour later. At this time the patient was still in coma. The pulse rate was 90, temperature was 100° and respiration 25 and shallow. The limbs were not spastic, and on raising and releasing them those on the right side fell more heavily than those on the left. The knee and arm reflexes were equal and sluggish, the plantar reflexes were absent. The pupils were equal and small, there were spontaneous inco-ordinate movements of the eyes. There was a bruise on the left side of the head in the temporal region. Gradually the man's condition became worse, his pulse rose from 90 till uncountable, his blood pressure fell, breathing became irregular, then stertorous, and the temperature rose to 105°. Within twelve hours of the injury he was dead.

A post-mortem examination showed—

(a) Hæmatoma in the left temporal muscle.

(b) Fracture of squama of the left temporal bone.

(c) Extradural hæmorrhage of medium dimensions.

(d) Bilateral subdural hæmorrhage.

(e) Extensive laceration of both temporal lobes with subarachnoid bleeding.

Comment.—Without a latent interval it is difficult to diagnose a surface hæmorrhage. The only neurological sign in the above case to point to surface compression was difference in tone in the *two sides of the body*, as shown by the test of the falling limbs. The bruise on the left side of the head was suggestive of fracture

it is to find the source of the bleeding, and much experience is necessary to control it. Posterior bleeding often comes from dural sinuses or from the veins which drain into them

SUBDURAL HÆMORRHAGES

I (i) Acute—*Diffuse*—A man aged thirty five years received an injury to his head as a result of a motor-car accident He was rendered unconscious immediately, and when admitted to hospital was in semicoma When first seen his face was pale and his skin cold and goose-fleshy It was obvious that apart from concussion he was also suffering from shock He was carefully undressed and put to bed in a warm room and left undisturbed for two hours

Depth of Unconsciousness—At the end of this period the man was not mentally accessible and could not be made to co-operate even in a simple examination He was restless and moved about the bed in a lurching way He reacted to painful stimuli and resented interference His condition was that of semicoma

Eyes—On opening the eyelids the gaze was vacant and the eyes wandered slowly but co-ordinately The pupils were equal, of mid dilatation, and reacted to light.

Power in Limbs—There were no paralyses

Reflexes—

Knee jerks	Active and equal (difficult to elicit)
Ankle jerks	
Abdominal	Present in all segments
Biceps	Difficult to elicit.
Triceps	
Supinator	
Plantar	Doubtfully extensor
Corneal	Active.
Swallowing	Present

Vomiting—There were occasional retchings with vomitings of small quantities of blood stained fluid When this happened the patient would strain and either turn on his side or sit forward No attempt was made to use a receiver placed in position by an attendant

Hæmorrhages—The nares were filled with blood, there was a little bleeding from the left ear Later a bilateral subconjunctival hæmorrhage developed

Records of the pulse rate, etc., will be found on the accompanying chart

Twelve hours after receipt of the injury there was no demonstrable

change in the depth of unconsciousness or in other neurological signs. Lumbar puncture in the left lateral position showed the cerebrospinal fluid pressure to be 220 mm. Blood-stained fluid was withdrawn containing 75,000 red blood corpuscles per cubic centimetre.

Twenty-four hours after receipt of the injury there was still no change in the neurological picture, save that when the man lurched from one position to another he did so with more of a writhe and tended to pull himself upright. These were perhaps signs of retrogression. Lumbar puncture in the left lateral position showed the cerebrospinal fluid pressure to be 180 mm. Blood-stained fluid was withdrawn containing 50,000 red blood corpuscles per cubic centimetre.

Radiography did not reveal a fracture of the skull.

Here, then, was a typical case of concussion associated with a mild subarachnoid hæmorrhage. The man's life was in the balance, and on the clinical evidence it was impossible at this stage to know whether he was going to succumb or recover. As judged by lumbar puncture and cell counts, the subarachnoid hæmorrhage had ceased.

Thirty-six hours after receipt of the injury there was little change in the neurological picture save that both plantar reflexes were probably extensor. On the other hand there was a definite deterioration of the pulse—its rate had increased and its volume fallen. The diastolic blood pressure had fallen appreciably (see chart). Lumbar puncture in the left lateral position showed the cerebrospinal fluid pressure to be 150 mm. Blood-stained fluid was withdrawn containing 30,000 red blood corpuscles per cubic centimetre.

Forty-eight hours after receipt of the injury the man's condition had deteriorated in every way. His pulse was faster and weaker. The respirations were increased and both systolic and diastolic blood pressures were falling. All reflexes were depressed; the pupils were dilated and the light reflexes were sluggish. The man did not react to painful stimuli and lurching had ceased. The jaw was lax and the man's face was bluish even when the jaw was held forward or when he was turned on to his side. The bladder was distended and micturition had ceased. From now onwards there was a gradual retrogression and the man died about sixty-four hours after receipt of the injury (Fig. 115).

Comment.—The man held his own for twenty-four to thirty-six hours and then for some reason succumbed to his injury. The retrogression was not due to a continuing subarachnoid hæmorrhage[1] Possibly it might be suggested that in some way

[1] Jefferson, G "The Nature of Concussion" *Brit Med Jour*, 1944, 1, 1

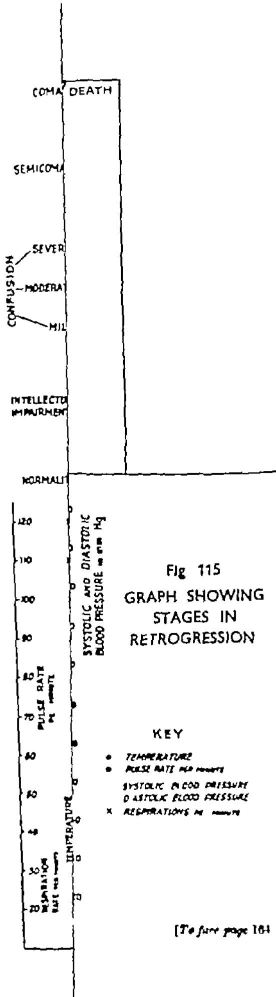


Fig 115
GRAPH SHOWING STAGES IN RETROGRESSION

[To face page 164

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[1] Jefferson, G "The Natur

a diffuse neuronal injury had become progressive, as suggested by Jefferson That is, an injury had set up a rapidly advancing degenerative encephalopathy At post mortem examination a small laceration was found on the under surface of the left temporal lobe and there was evidence of a moderate subarachnoid hæmorrhage, red blood corpuscles filling the numerous sulci of the brain No large clots were found in the cortical spaces or basal cisterns There were scattered petechial hæmorrhages, particularly in the region of the laceration Both subdural spaces were filled with a considerable amount of liquid blood It can be reasonably suggested that it was the bilateral subdural hæmorrhage which turned the scale The objection that the cerebrospinal fluid pressure should have risen had this been the case does not invalidate the possibility, because rapid compression of the brain does not necessarily cause a rise in cerebrospinal fluid pressure No doubt the best treatment in this case would have been to drain the subdural space on both sides through trephine or burr holes

Botterell,[1] Dott, Alexander and Ascroft[2] suggest that in unilateral cases of acute subdural hæmorrhage the subdural space should be opened widely either through a decompression or through a bone flap exposure

It must be realised that, as a rule, there is no clinical syndrome which can be attributed to acute subdural hæmorrhage The picture of the diffuse neuronal injury is so dominant that subdural hæmorrhages can colour it only when they are severe and extensive

I. (ii) Acute—*Localised*—A young woman aged thirty one years was knocked down by a tramcar in such a way that she struck her head on the ground when she fell She was rendered unconscious immediately On arrival at an outside hospital her mental state was found to be that of confusion The left pupil was oval in shape and reacted sluggishly to light. The right pupil was small and its light reactions were also sluggish There were no other neurological signs. Three hours later the left pupil became widely dilated and fixed and both plantar responses became extensor in type. By this time the pulse rate had dropped from 80 to 64 beats per minute. At this stage the patient was transferred to the neurosurgical unit. Here the following points were noted —

(*a*) There was a large bruise in the left temporal region
(*b*) The state of unconsciousness was that of semicoma
(*c*) The left pupil was fixed and widely dilated
(*d*) The left upper eyelid was drooped

[1] Paper read at Meeting of British Neurosurgical Society July 1942.
[2] Chapter LX "Surgery of Modern Warfare" Third Edition (1944). E. & S. Livingstone Ltd., Edinburgh.

(*e*) All four limbs were spastic but could be made to move by suitably applied painful stimuli

(*f*) Both plantar reflexes were extensor in type.

A diagnosis was made of a surface clot, and because of the left fixed pupil was lateralised to the left side.

Operation (*N.W.*).—In the first instance two inspection burr holes were made over the left temporal region Although no extradural clot was present, the dark colour of a subdural collection of blood could be plainly seen. On incising the dura mater a large gelatinous mass of blood was found. As satisfactory removal of the clot was impossible through the small inspection holes a suitable osteoplastic flap was raised; this revealed quite clearly the boundaries of a large clot which, in its thickest depth, was $1\frac{1}{2}$ in The underlying brain was bruised and swollen After the clot had been removed a decompression was provided, the subdural space drained and the wound closed in layers. The next day the patient's mental and general condition had improved and after about a week she was fully conscious At this time it was discovered that she was suffering from a nominal aphasia. This speech difficulty improved as the weeks went by, and she finally made a complete recovery.

This is a rare example of a localised subdural hæmatoma which was amenable to surgical treatment and which was associated with a non-lethal injury of the intrinsic type.[1]

II. Chronic—(1) *Early*.—On 8th May 1943 a soldier aged thirty-three years was struck in the face by another man's fist during an argument and fell to the ground. He was dazed, but according to witnesses was not rendered unconscious. Shortly afterwards he was taken to the police station where he was found to be abusive and excited. He was returned to his unit and nothing relevant occurred until 10th May 1943. On this day, while doing P.T exercises, he collapsed and became unconscious, whereupon he was taken to the nearest hospital. There he was found to be drowsy, with a pulse rate of 48. On 11th May 1943 he was transferred to the head centre, in no worse condition after a journey of sixty miles.

On admission he was a little drowsy and confused but could easily be roused to co-operate in a detailed neurological examination. He complained of a severe generalised headache which was aggravated by movement. Apart from drowsiness there were no other abnormal neurological signs. There was a small abrasion over the left eye and on the left ear, but no bruising or swelling on the scalp. The pulse rate varied from 42 to 56, the

[1] N Whalley "Acute Subdural Hæmatoma Amenable to Surgical Treatment" *Lancet* 7th February 1948, p 213

blood pressure being 130/80 Nothing abnormal was found in the urine X ray proved that the skull had not been fractured In the lateral position the cerebrospinal fluid pressure was 120 mm , which is normal No blood nor any other abnormality was found in the fluid

On 17th May 1943 the patient complained of exceedingly severe headache and nursed his head in his hands Again he was co-operative and there were no abnormal neurological signs. The cerebrospinal fluid pressure on lumbar manometry was 120 mm , and withdrawal of 20 c.c. gave him considerable relief from the head pains The pulse rate varied from 40 to 50 On 21st May 1943 he was difficult to rouse, but there were still no localising signs, apart from the disturbance of consciousness , the pulse was steady at about 40

In view of the persistency of the headache, which was described as a severe pain and not an ache, the slow pulse and the deepen ing unconsciousness, a diagnosis was made of a subdural hæmor rhage. Under local anæsthesia two burr holes were made 3 cm from the midline, 10 cm above the external occipital protuberance A large fluid hæmatoma was found in the subdural space on the right side This was evacuated by suction and the resulting cavity drained for forty eight hours Immediately after evacuation of the clot the man volunteered the information that for the first time since he had been in hospital his pain had been relieved Convalescence was uneventful, and he was transferred to a rehabili tation centre on 9th June 1943 When he left hospital his pulse rate was 65

Comment —This is a typical case of so-called chronic subdural hæmatoma. Following a mild injury to the head, when severe pain and not an ache is complained of, the possibility of a subdural hæmorrhage should be considered There may be no sign of raised intracranial pressure, as shown by the optic discs and by mano- metry and there may be no localising neurological signs In creasingly severe headaches, followed by the slightest impairment of consciousness, are extremely suggestive of a chronic subdural hæmatoma. Owing to the frequency of their bilaterality both sides of the head should be always explored when a subdural hæmatoma is suspected

Bradycardia without impairment of consciousness or severe headaches is not of any particular pathological significance

(ii) *Late* —This description is concerned with the case of a boy aged thirteen years who developed a severe headache which at first rapidly receded but later became persistent The pain started in the nape of the neck radiated upwards chiefly through the left side of the head and was of a severe aching character

Vomiting was frequent and not related to anything in particular save that it was apt to be precipitated by movements, such as bending. The boy had been ill for six weeks, but symptoms other than headaches and vomiting had not developed Apart from the usual blows that a schoolboy is apt to receive to his head, there was no history of concussion

When first admitted to the University Hospital he was drowsy, but could easily be roused to co-operate in examination. The power of the limbs was not impaired, and there were no abnormalities in the reflexes to indicate a lesion of the pyramidal pathways. There was no sensory loss. Co-ordination of movement was smooth; both pupils were widely dilated but reacted to light and accommodation. The disc margins were indistinct but there was no measurable degree of papillœdema. There was a slight bilateral external strabismus which did not cause diplopia The pulse rate was 48, the blood pressure was 100/55. The intracranial pressure, as measured by manometry in the lateral position, was 300 mm. cerebrospinal fluid. There were no cytological or chemical abnormalities in the fluid.

Within a few days of his admission a measurable degree of papillœdema had developed, as well as a left facial weakness, diplopia on looking to both sides, and a fine nystagmus on lateral fixation of the eyes. A diagnosis was made of an expanding intracranial lesion. whereupon the boy was transferred to the neurosurgical unit.

Here he was found to be a little sleepy but could easily be persuaded to co-operate in a detailed neurological examination to the extent of walking unaided. Neurologically there was a mixture of signs referable both to the cerebellum and to the cerebral hemispheres, associated with those of raised generalised intracranial pressure. The skull was large and asymmetrical, there being a definite ballooning in the left temporal region. At some periods of the day the boy would be awake. at others he was difficult to rouse.

There was in this case no guide in the history to point to the diagnosis of a chronic subdural hæmatoma, and this is quite common. In many cases the information comes out only in retrospect An expanding lesion was all that could be diagnosed. The ballooning of the skull may have been due to a hydrocephalus, but usually a distinct local bulge is due to an underlying lesion, and in my experience the commonest of these are cysts, surface gliomas and cholesteatomas

Two burr holes were made in the skull under local anæsthesia, 3 cm. from the middle line and 8 cm above the external occipital protuberance with the object of making a ventriculogram.

A large cystic hæmatoma was found on the left side and

evacuated. As the clot was large, an anterior counter opening was made to ensure thorough drainage and to allow the clot to be washed out. The boy made a complete recovery.

Comment.—In a long standing chronic subdural hæmatoma, as this one must have been, judged by the changes in the skull, the possibility of injury being a causative factor is often forgotten by the patient. Reliable localising or lateralising signs are often absent. The outstanding feature is transient losses of consciousness with unaccountable periods of recovery.

III. Subdural Hygromas.—A boy aged eighteen years, while riding his bicycle, crashed; he remembers falling and hitting his head against the ground. He picked up his bicycle and wheeled it home, where on account of the severity of his headache he went to bed and slept. The next morning his mother had difficulty in rousing him. When awake he complained of severe generalised headaches. A large bruise had formed on the front of the right side of the head. He stayed away from work, and on the fourth day following the injury was sent to hospital because of increasingly severe headaches. When left undisturbed he would go off into a deep sleep although he could easily be roused and persuaded to co-operate. There were no localising or lateralising neurological signs. There was no papillœdema. The pulse rate was 42. In the left lateral position the intracranial pressure, as measured by lumbar manometry, was 200 mm. of cerebrospinal fluid, which is a little higher than normal. The cerebrospinal fluid chemistry and cytology were normal. There was a large swelling on the right side of the head, due obviously to a subgaleal hæmatoma.

On the sixth day following injury the neurological state was much the same, save that an indefinite plantar extensor response had developed on the left side. The pulse rate was 54, temperature, respiration and blood pressure being normal. Radiography proved that the skull had not been fractured. A diagnosis of a subdural hæmatoma was made.

At operation a trephine hole was made on the right side of the skull. As the left extensor plantar response was the only localising sign, the hole was placed over the right motor cortex. On making a cruciate incision into the dura, 15 c.c. of yellowish fluid were tapped and drained. Immediately the boy was relieved of his head pains, drowsiness disappeared, and within ten days he went back to work and has been well ever since.

SUBARACHNOID HÆMORRHAGES

Acute subarachnoid hæmorrhage in association with other forms of cerebral trauma, particularly of the diffuse neuronal

type, occurs in at least 75 per cent. of cases of severe injury to the head. As an isolated occurrence it is rare. In the typical case there is no distinct clinical picture which can be attributed to subarachnoid hæmorrhage, although probably chemical meningeal irritation is the main cause of the restlessness and unruliness which are so common and troublesome a feature of cerebral trauma.[1]

In spontaneous rupture of congenital aneurysms severe subarachnoid bleeding usually occurs without impairment of consciousness. In head injuries, on the other hand, the functions of the brain are already seriously embarrassed. Therefore loss of blood, which may be considerable in subarachnoid bleeding, associated with the shock of pain and interference with the circulation of the cerebrospinal fluid by extravasated red blood corpuscles, may seriously jeopardise the chances of recovery.

Early Delayed Hæmorrhage.—A man aged thirty-four years, while riding home one evening from work on his bicycle, skidded on the tram lines and fell, hurting his head. He was concussed for a time but was fully conscious within a few hours and went to bed and slept. The following morning he was reasonably comfortable but in the afternoon he began to complain of severe occipital head pains which soon became intolerable, and morphia had to be injected to give him relief.

When admitted to hospital the man was fully conscious and fully co-operative, but was loath to make any kind of movement because of the discomfort this caused him He complained of intolerable pains in the whole of the head and neck. It was painful for him even to open his eyes. Apart from neck stiffness there were no abnormal neurological signs Lumbar puncture proved the cerebrospinal fluid to be loaded heavily with blood to the degree of 300,000 red blood cells per cubic centimetre of cerebrospinal fluid After repeated lumbar drainages of cerebrospinal fluid, which gave him considerable relief, he finally made a satisfactory recovery, although for many months he complained of residual headaches.

Comment—There is a possibility in this case that the hæmorrhage was due to spontaneous rupture of a congenital aneurysm and that the original fall from the bicycle was not accidental Possibly the blow on the head caused by an accidental fall caused the rupture of a congenital aneurysm

Alternatively, a congenital aneurysm may not have been present What is certain is that a severe subarachnoid bleeding occurred twelve hours after a blow on the head, and this might

[1] Sartorius, K "Focal Cerebral Injury the Type of Injury to the Brain following Assault" *Clinical Proc*, 1947, 6, No 6

reasonably be attributed to the blow *Previous to the accident* there had been no evidence of an organic intracranial lesion

Late Delayed Hæmorrhage—A man aged fifty five years was admitted to hospital suffering from a severe subarachnoid hæmorrhage, as proved by lumbar puncture. He complained of severe pains in the head and of a stiff neck He was fully conscious and fully co-operative The history was that while ascending some stairs he was struck in the back of the head by a violent pain. Things went black before his eyes for a few minutes and he fell on to his knees *There was no injury in this instance*, the hæmorrhage being purely spontaneous It was discovered that six months previously the man had received a severe injury to his head which had rendered him unconscious for nearly two days The fact that he had received an injury to his head at the first episode was confirmed beyond doubt. Before his accident he had been a perfectly healthy man, as proved by his medical and working records *It is not unreasonable to assume, in this case,* that at the time of accident a surface vessel was contused and that this led to weakness of its walls and to late delayed hæmorrhage

INTRACEREBRAL HÆMORRHAGE

Diffusely scattered petechial hæmorrhages in the brain substance are common and no clear cut clinical picture can be attributed to them Massive intracerebral hæmorrhages, on the other hand, are relatively rare and are usually due either to surface laceration, with tearing of a cortical vessel which bleeds into the substance of the brain, or to delayed post traumatic apoplexy

MASSIVE INTRACEREBRAL HÆMORRHAGES

I **Acute**—A middle-aged man was struck in the small of the back by *the radiator of a motor car He was thrown into the air* in a somersault and crashed on to the road head downwards In this accident the brain was subjected to the forces of acceleration deceleration and crushing the details of the accident being witnessed by the author When examined a few seconds after his injury the man was found to be deeply unconscious, his face was pale and his pulse weak He was carried into a near by house *and kept under continuous observation for two hours* He was in coma and the corneal reflexes were absent All the muscles were flaccid and the limb reflexes were absent When the jaw was released the tongue fell backwards and obstructed breathing Both pupils were fully dilated and fixed there were no movements of the eyes Breathing at first was regular fast and shallow,

later it became gasping and then of the Cheyne-Stokes type The pulse from the first was fast and weak, gradually increasing in rate and decreasing in volume until after an hour it was uncountable and almost impalpable. In three hours the man was dead

A post-mortem examination showed .—

(a) A large and deep laceration on the under surface of the right frontal lobe

(b) A large hæmorrhage in the frontal lobe of the brain impinging on the hypothalamus.

(c) A diffuse subarachnoid hæmorrhage.

(d) Diffusely scattered petechial hæmorrhages.

Comment.—Massive hæmorrhages which develop immediately after injury indicate that the brain has been subjected to injurious forces of overwhelming magnitude. It is likely, therefore, that the clinical picture in the above case was due partly to diffuse neuronal injury and partly to the intracerebral hæmorrhage, each lesion in itself being severe enough to be fatal.

II. Delayed—(1) *Early*—At 3.30 P.M on 24th December 1943 a man aged fifty-six years was struck on the face, lost his balance and fell backwards, striking his head heavily on a stone step. He was dazed for a few minutes but soon regained consciousness sufficiently to return home. An hour later he became drowsy. The next day he was admitted to hospital. He was unconscious but could be made to give occasional monosyllabic answers. He was spontaneously restless and resented any kind of examination. Lumbar puncture revealed a blood-stained cerebrospinal fluid. At this stage the pulse rate was 80 and temperature 101°. Gradually unconsciousness deepened and the pulse rate rose to 120 and the temperature to 103°.

On 26th December 1943, at 8.30 P.M., he was admitted to the neurosurgical unit, when he was found to be in coma and could not be roused either by noise or by painful stimulation The corneal reflexes were present. All the limbs were spastic, the right ones being more so than the left The knee, ankle, arm and abdominal reflexes were all absent The right plantar reflex was extensor, the left being doubtful. The pupils were small and reacted to light The eyes moved conjugately. The optic nerve heads were not œdematous. The bladder was empty The temperature was 101°, the pulse rate 82. Respiration was fast and stertorous and the blood pressure 170/110 There was slight bruising about the left orbit. There was no bleeding from the nose or ears. X-rays showed a linear fracture of the vault above and behind the right ear.

Here was a case of a head injury with a lucid interval In

view of the temperature the possibility of a fulminating meningo-encephalitis had to be considered Against this diagnosis was the early onset of drowsiness and the absence of pus in the cerebro-spinal fluid twenty four hours after the accident A diagnosis of a massive hæmorrhage was made on the history of the lucid interval

At operation bilateral burr holes were made to eliminate the possibility of an extradural hæmorrhage but none was found On opening the subdural space a bilateral subdural hæmorrhage was found but not of degree sufficient to account for the clinical signs A left sided temporal decompression was made, when the *brain was found to be under great tension.* The left side was chosen for decompression because the neurological signs were a little more *marked on the right side of the body* Slowly the man's condition deteriorated and he died at 6 40 A M on 27th December 1943

A post mortem examination showed —

(*a*) A small undisplaced linear fracture along the line of the right occipitoparietal suture
(*b*) A large hæmorrhage in the right frontal lobe of the brain.
(*c*) A lesser hæmorrhage in the posterior end of the left temporal lobe.
(*d*) A subdural and subarachnoid hæmorrhage of small degree

Comment —Here was a case in many ways indistinguishable from that of an extradural hæmorrhage In fact it would have been justifiable to make such a diagnosis. The features against an extradural compression were —

(*a*) In cases of extradural compression a rise of temperature is usually a terminal and not a relatively early event, as it was in the above case
(*b*) Restlessness is unusual in extradural or subdural com pression, whereas in apoplexy patients often go into coma noisily
(*c*) In the early stages of extradural compression there are often frank localising neurological signs There were few in the above case

(II) *Late* —A man aged thirty five years, a joiner by trade, received an injury to his head while at work early in December 1940. According to his own evidence he was struck on the *top* of the front part of the head by a baton or a falling piece of timber He was dazed for a time the pain being considerable. He sat down for a few minutes and later was able to continue with his

work. That same night he complained of headaches and described the incident to his wife. He returned to work the next day, and towards the end of the month he received another blow on the head by a piece of timber, two of his colleagues witnessing the accident. He was badly dazed but was never unconscious From this time onwards he complained of repeated headaches.

On 2nd January 1941, while getting out of bed, he became dizzy and fell to the floor. On coming to his aid his wife found that the left side of his body was completely paralysed Within an hour the man was in semicoma, and it was in this state that he was admitted to hospital. A diagnosis was made of an intracerebral hæmorrhage. Lumbar puncture proved that there had not been any subarachnoid bleeding.

The man was fully conscious within a week, but was left with a complete and dense left-sided hemiplegia. By 19th March 1941 the neurological condition had not changed. On this date the man was transferred to the neurosurgical unit On admission he was fully conscious and co-operative. There was a complete left-sided hemiplegia without associated sensory loss. The optic nerve heads were flat and the cerebrospinal fluid pressure, as measured by lumbar manometry, was normal The chemistry and cytology of the cerebrospinal fluid were normal.

Professor Hume reported that there was no evidence of generalised cardiovascular disease, the blood pressure being normal.

There was no evidence of kidney or of any other metabolic disease. The Wassermann reaction in the blood and the cerebrospinal fluid was negative. The man's health previous to the accident was proved to have been satisfactory. He did not suffer from headaches and had never had a fainting attack. A diagnosis of a right-sided intracerebral hæmorrhage was made.

On 24th April 1941 an encephalogram was performed, 50 c c. of cerebrospinal fluid being withdrawn and 50 c.c of oxygen injected. The gas studies revealed a filling defect in the right lateral ventricle This finding confirmed the diagnosis of an intracerebral hæmorrhage.

After rehabilitation the man was able to limp about on sticks, but a short time later his general and mental health began to deteriorate, and in about six months he was virtually bedridden.

On 13th April 1942 he was readmitted to the neurosurgical unit. He had lost several stones in weight and was obviously a very ill man. He was confused; his attention easily flagged, and he was imperfectly oriented He was extremely emotional and often burst into tears The left side of the body was paralysed and spastic; the right side of the body was weak and movements were tremulous. The optic nerve heads were pale but flat. Lumbar puncture proved the cerebrospinal fluid to be under no increased

pressure Nothing abnormal was found in the fluid The Wassermann reaction in the blood and cerebrospinal fluid was again negative Encephalography revealed a bilateral internal hydrocephalus, the wastage being most marked on the right side of the brain

The man died on 23rd May 1942 and a full post mortem examination was performed

No disease was found in the abdominal or thoracic organs. In the brain a congenital aneurysm was *not* found on any of the large intracranial vessels, and particularly on those forming the circle of Willis The lumen of the extracerebral part of the middle cerebral artery was patent There was slight nodular thickening of the intima and some reduplication of the internal elastic lamina, the appearances indicating a mild grade of nodular atheroma In the areas forming the boundaries of the right Sylvian fissure, for a distance extending 10 cm vertically and anteroposteriorly, the brain tissue was shrunken and depressed the pia arachnoid over the affected parts being shrunken and adherent, while the brain tissue itself was pale and brownish in colour On section the grey matter all round the right Sylvian fissure had quite disappeared and consisted of greyish tissue in which there were minute cysts The whole of the right internal capsule was shrunken and quite hard, as was the optic thalamus and lenticular nucleus The grey matter was replaced by gliosis There was no direct evidence of a residual hæmatoma

Comment—As Symonds [1] has pointed out the occurrence of delayed intracerebral hæmorrhage resulting from injury to the head has been recognised since Bollinger [2] wrote on this subject in 1891 According to Bollinger, the causal pathology is that at the time of injury an area of brain is contused This leads to a focus of softening Here the wall of an artery is occasionally involved Necrosis occurs, with rupture and hæmorrhage

If this be the true explanation of delayed post traumatic intracerebral hæmorrhage, then it can readily be understood how minor injuries to the head can lead to serious results

Delayed post traumatic hæmorrhages fortunately, are a rare sequel of head injury Moreover, they are apt to be confused with those hæmorrhages which result from the spontaneous rupture of congenital aneurysms Such aneurysms are developmental in origin and occur chiefly on the large vessels forming the circle of Willis They are not the result of syphilis or of raised blood pressure Usually they burst into the subarachnoid space but

[1] Symonds, C. P "Delayed Traumatic Intracerebral Hæmorrhage *Brit Med Jour.*, 1940, 1, 1048.

[2] Bollinger O "Internat Beiträge zur wiss Med. Festschrift Rud Virchow" Berlin, 1891

occasionally rupture into the intracerebral tissues with resulting intracerebral hæmorrhage

Symonds has described a case of a chronic intracerebral hæmatoma. In this case it was presumed that an injury to the head led to an intracerebral cyst. A later injury caused a secondary hæmorrhage into the cyst and compression of the brain. Such chronic intracerebral hæmatomata can occur from spontaneous ruptures of cerebral vessels, and two examples occurred in the author's series

From the medico-legal point of view the evidence produced by various observers and that of the above case testifies to the ætiology of trauma in some cases of apoplexy.

INFECTIONS

Early.—A man aged twenty years received an injury to his head on 23rd March 1943 as the result of a motor-cycle accident. He was knocked unconscious and was conveyed to hospital in this state Three hours after the accident he recovered consciousness sufficiently to co-operate usefully in a neurological examination, although he was very restless and resented being disturbed There were no clinical signs to point to localised damage in the brain. The forehead was swollen. There was profuse nose bleeding but little subconjunctival staining. The following day the man was very much better, he was fully co-operative and less restless, the temperature being normal Radiography showed a fracture of the nasal process of the left superior maxilla

On the evening of 26th March 1943 there was a sudden rise of temperature to 101° F, when the man became restless and complained of severe generalised headaches Two hours later he was obviously worse, his temperature was 102 5° F, the pulse rate 100 per minute, and respiration 30 per minute. He was drowsy, restless and in pain, as judged by the way he held his head in his hands. The neck was stiff, and Kernig's sign was positive.

A diagnosis of meningitis was made and confirmed by lumbar puncture. A full course of sulphonamide therapy was given. By 28th March 1943 the man was greatly improved. He was fully conscious and co-operative but a little slow in responding to questions and acceding to requests He complained of headaches Apart from his mental sluggishness there were no abnormal neurological signs The temperature was normal and the cerebrospinal fluid was clear and not under pressure. His nose was blocked by congealed blood but there was no discharge of cerebrospinal fluid either from the nares or into the nasopharynx. He was

warned not to blow his nose. His forehead was still swollen Further X rays showed the frontal air sinuses to be opaque, presumably with extravasated blood, but no fracture of the vault or base of the skull was demonstrable even by stereoscopic and special views

On 1st April 1943 his temperature rose to 100° F and he complained again of severe headaches His neck became stiff, and again there were obvious clinical signs of meningitis. The cerebro spinal fluid pressure was raised to 270 mm, the fluid being opaque and loaded with pus cells The causative organism was proved to be a pneumococcus In spite of serum and sulphonamide therapy the man's condition slowly deteriorated and he died on 9th April 1943

A post mortem examination showed —

(a) The frontal and ethmoidal air sinuses were filled with blood

(b) There was a thin layer of extradural blood on the floor of the anterior fossa and behind the vertical posterior walls of the frontal air sinuses.

(c) There was a linear fracture of the right side of the anterior fossa extending into the ethmoidal air cells.

(d) The dura mater was intact

(e) There was a superficial laceration on the under surface of the left frontal lobe

(f) There was a diffuse meningitis

Comment —This is a typical picture of a severe fronto-ethmoidal injury Such injuries are usually caused by a blow over the forehead, and particularly over the root of the nose The man concerned is concussed and may or may not recover from his concussion In the above case he did recover consciousness At the time of accident a fracture of the skull occurs which is internally compound and which, for technical reasons is extremely difficult to demonstrate by radiography In my experience leakage of cerebrospinal fluid is usually masked by bleeding and passes undetected even though it may be suspected As seen in the case above meningitis can occur without laceration of the dura mater and cerebrospinal fluid leakage

Whether closed paranasal sinus fractures should be treated conservatively or whether the cribriform plates and posterior walls of the frontal air sinuses should be explored surgically as a routine to prevent meningitis has not yet been decided My custom has been to be conservative in all cases where the concussional element is severe Sulphonamide therapy of course should be given as a routine prophylactic measure Open

exploration for possible repair of torn dura is indicated when there is undoubted evidence of cerebrospinal fluid leakage or when radiography shows a badly displaced fracture of the walls of the ethmoidal and frontal air sinuses which form the inner table of the anterior fossa.

Late.—A man aged thirty-nine years received a severe injury to his head as a result of a blow with a spade When admitted to hospital he was found to be semiconscious. His forehead was swollen and bruised, and there was profuse bleeding from the nose. On the left side there was an extensive subconjunctival hæmorrhage The man did not regain consciousness until the third day. By the end of the third week he was walking about, the swelling on the forehead had disappeared but there was still evidence of subconjunctival hæmorrhage He was allowed to leave hospital, but on his wife's evidence he never made a complete recovery. He was slow, moody and often confused.

Many weeks later he was admitted to the neurosurgical centre because of headaches and strangeness of manner

On examination the man was obviously confused. He realised that he had received a head injury but found it difficult to understand why he was in hospital His memory was poor; concentration was faulty and he was imperfectly oriented regarding time and place. He understood the spoken word but found speech difficult, not easily finding the right words to express his thoughts. He was continent Apart from his mental confusion and obvious motor aphasia there were no other abnormal neurological signs. The optic nerve heads were flat. The temperature, blood pressure, pulse rate and respiration were normal. Radiography revealed an extensive fracture of the skull with the fracture running into the left frontal air sinus. Both walls of the frontal air sinus were broken into numerous fragments with slight backwards displacements.

Shortly after admission he complained of a very severe headache, when his temperature was found to be raised to 100° F. In two hours he was stuporose, when lumbar puncture proved him to be suffering from a diffuse pyococcal meningitis. Within eighteen hours of the rise of temperature he was dead.

A post-mortem examination showed :—

(*a*) A comminuted fracture of the left frontal air sinus.
(*b*) Pus in the frontal air sinus.
(*c*) Infected granulation tissue matting the dura to the posterior wall of the frontal air sinus
(*d*) A fistula passing from the frontal air sinus through the dura into the brain.

(e) A chronic abscess in the left frontal lobe of the brain about the size of a pigeon's egg which had not ruptured into the ventricle.
(f) Diffuse cerebral œdema.
(g) Diffuse meningitis

Comment—Chronic cerebral abscess formation following closed head injuries is rare There had, of course, been internal compounding due to fracture of the frontal air sinus in the above case The man's mental signs during the period of convalescence had no doubt been due to the formation of the frontal intracerebral abscess His terminal illness had been due to a leakage of infection and diffuse meningitis

In another case a man died from a spreading subdural abscess following an acute subdural hæmorrhage

DIFFUSE NEURONAL INJURIES

In the early hours of the morning of 22nd January 1944 a girl aged twenty two years fell from her pedal cycle and injured her head When picked up she was unconscious but could be roused to some extent, although not sufficiently to speak or to co-operate She was bleeding from the nose and there was a large abrasion over the left forehead She moved both arms and legs freely The pupils were equal and reacted freely to light. After being admitted to a first aid station she vomited a mixture of old and new blood She was incontinent of urine The pulse was strong and respiration regular and deep Twelve hours later she was admitted to the neurosurgical unit.

Depth of Unconsciousness.—At this time she was in semi coma She lay still, in a supine position, with her head resting on one side She could not be made to speak or to obey simple commands but reacted to simple painful stimuli

Eyes—On opening the eyelids the eyes looked forwards in a vacant stare and occasionally moved from side to side co-ordinately The pupils were equal of mid dilatation and reacted briskly to light

Power in the Limbs—There were no paralyses

Reflexes—

Knee jerks Ankle jerks	Active and equal
Abdominal	Flicker in all segments
Biceps Triceps Supinator	Active and equal

Plantar . . .	Both extensor.
Corneal .	Active.
Swallowing .	Present.

Vomiting.—There was repeated vomiting of small quantities of blood-stained bile.

Hæmorrhages.—The nares were filled with coagulated blood. On the left side there was a small subconjunctival hæmorrhage which later became extensive. A hæmatoma was present over the left forehead

Pulse.—92 per minute.

Temperature.—98 4° F.

Respiration.—20 per minute.

Blood Pressure.—110/70 mm. Hg

X-rays.—There was no radiographic evidence of fracture of the skull.

Here, presumably, was a case of a diffuse neuronal injury As far as could be judged, the girl was rendered unconscious immediately on receipt of the injury, and after twelve hours showed no sign of recovery. From the neurological evidence it was impossible to know whether the girl was going to recover or to succumb from her injury. The chances, as judged by previous experience, were four to one that she would recover. Her life, however, was in the balance

Neurological observations were made at frequent intervals.

Stages in Recovery.—On the morning of 23rd January 1944 there was evidence of improvement, as shown by the return of spontaneous movement and the adoption of a more normal posture instead of an apparently uncomfortable sprawl across the bed. In the afternoon of this day the girl would occasionally open her eyes, and by her gestures it was obvious that she recognised her parents.

In the evening she reacted in a more purposive way to painful stimuli; in particular she would attempt to remove the offending stimulus with a sweep of her hand rather than escape by a vigorous lurch into another position. She was still doubly incontinent. She made no request for drinks or food but swallowed adequate quantities of milk and sugared water with little resistance or difficulty when fed by the nursing staff. As subsequent events proved, the period of semicoma was coming to an end, and the next day the girl had reached the state of severe *confusion*.

On the morning of 24th January 1944 she began to open her eyes and look around the ward. She asked for a bed pan and used it, but on two occasions wet the bed. Also, she asked for drinks and swallowed semi-solids such as custard without difficulty.

She spoke to her parents in short snatches, but would soon curl up on her side and apparently go to sleep

On the fifth day after the accident she was fully continent. She attempted to converse with the nursing staff but apart from simple requests, was unable to carry on a sensible conversation. The plantar reflexes for the first time were definitely flexor

From this period onwards improvement was noticeable and gradual The girl began to show sustained interest in her environment and began to sit up and ask sensible questions.

On the ninth day after injury the following conversation was recorded —

Question.	*Answer*
Good morning	Were you playing about with me a short while ago ? (She had been examined by an assistant shortly before)
No I was not	I think this is complicated I have only your word for that
Do you know why you are here ?	I know that I was stationed at (correct answer) but what I came here for I don t know I cannot remember any more but it all seems some time ago
Do you know your name ?	Yes. (Correct answer)
How old are you ?	Twenty-one
Do you know what happened to you ?	I don t know and that is why I am asking everybody There is something wrong with my head I know that I have never had this feeling before
Do you know where your are ?	I think I am near Carlisle (Actually she was in Newcastle-upon Tyne)
Are you in the Services ?	Yes
Which Service ?	(Correct answer)
What is your work ?	Clerk, general duties
What did you do before you joined the Services ?	I was an art student
What were you doing ?	Studying art
What kind of art ?	I was a model of a kind and also a model for dresses.
Were you taking your degree ?	No
Were you studying there ?	Yes for just over a couple of years
What were you going to do then ?	Get hold of somebody to marry I suppose—that is about the truth of it
Is that why you went to the University ?	It just happened that way
If you didn t marry what then ?	Keep on being a model until something did happen I suppose (smiled) Confessions of an artist s model at play

Question	*Answer*
Have you a headache ?	I don't know exactly, but there is something wrong with my head
Can you smell and taste ?	Yes
What are you going to do when you leave here ?	Go back, I suppose
Do you like your work ?	Gosh, of course I do
What kind of work do you do now ?	I am a shorthand typist

Two days later a further conversation was recorded as follows .—

Question	*Answer.*
Where are you now ?	Carlisle
Can you recognise anybody about you ?	I saw you yesterday
Who is this ? (Staff Nurse)	I remember having seen her before, but that is all
What about the other nurses ?	I do not remember anybody else
What day is it ?	Someone told me it was Saturday. (It was Monday)
What time is it ?	(Turning her head towards the ward clock) According to that clock it is three o'clock (Correct)
What is the date ?	Someone told me before
What year is it ?	January 1944, that I do know
How is your head feeling ?	It feels peculiar, as though it did not belong to me
Have you any pain ?	No pain whatever—just as though it was not my head
Can you sleep all right ?	I cannot remember having been asleep, but I must have been
What were you in civilian life ?	An art student
What did you do there ?	Attended the art school as an art student
What kind of art ?	Architectural—modelling
What do you mean by modelling ?	Drawing—I could at one time.
What does an art student mean ?	To draw for somebody
How do you like life in the Services ?	I like it very well indeed
Do you like it better than College ?	I like it very much, but that cannot go on unendingly
Do you want to go back to the Services ?	The sooner the better.

According to her mother, the girl had always been a strong healthy person who did very well at school. She left school at the age of sixteen and went to an art school for one year. At the approach of the war she was sent to a commercial college to learn shorthand and typewriting. She was there for about nine months, during which time she lived at home At the end of her training she obtained work as a shorthand typist She was

very anxious to join one of the Services, and when she eventually did so she thoroughly enjoyed the life

The mother stated that her daughter knew her but that she could not remember how many times she had visited her She also recognised a boy friend whom she had met for the first time a few weeks previously

In the subconscious phase she had been playing with a ring and occasionally had put it on her engagement finger

On 4th February 1944 a further conversation was recorded as follows —

Question	*Answer*
How are you getting on ?	I am getting on all right but I cannot function my eyes properly
Can you see properly ?	I can see farther every day—I can now see the end of the ward
Do you know where you are ?	Newcastle—I don t know where I got Carlisle from—some department O 3 and 4 or something (Correct) The hospital is called Westcliffe General Hospital
Do you know what happened to you ?	I know why I am here
Why are you in hospital ?	I remember something—I haven t been able to discover—I remember getting on a bicycle and fixing the lights—being at the Sergeants' Mess.
What were you doing at the Sergeants Mess—was it male or female ?	Mixed.
Can you think quite clearly now ?	Yes I can say that—there is some thing wrong with my head that makes it buzz at times
What is my name ?	I haven t been told.
What is Sister s name ?	I do not know anybody s name.
Read something out of the paper	(She read the paper correctly but in a staccato voice)
	Voluntarily she said that she had started to write letters but that she didn t know what they were like

In the last week before transfer to the rehabilitation centre the girl was satisfactorily co-operative and fully oriented regarding time and place Her mental processes, however, were far from normal There was a great tendency to childishness and silly facetiousness One of her amusements was to pretend not to recognise someone she knew perfectly well in order to embarrass them In many similar ways she was mildly impudent She was willing to describe the intimate details of her private life to anyone ready to listen On the other hand, she never carried

on a sustained sensible conversation with any one of the nursing staff. The impression she gave was that her mind was far away from her immediate environment and that she did not properly comprehend what had happened to her. She showed no sign whatever of depression.

On 13th March 1944, following a course of rehabilitation, there were no abnormal neurological signs, and the girl's only complaint was of difficulty in focusing the eyes on distant objects.

PROLONGED UNCONSCIOUSNESS AND AMNESIA

Occasionally a patient may remain in or near semicoma for two or several weeks. In my experience this type of patient usually dies if the causative condition cannot be relieved by dehydration or a simple operation. For many days there is no demonstrable change in the depth of unconsciousness nor any deterioration in the pulse, blood pressure, etc. Then, in spite of the introduction of adequate quantities of the correct type of food into the stomach, either by swallowing or by intubation, the patient begins to get thin and lose weight. This sign is often the beginning of the end. The usual sequence of events then sets in. The pulse rate increases, the blood pressure falls, respiration increases, the temperature rises and the muscles become flaccid and the reflexes disappear.

The underlying pathology in such cases is often obscure as the prolonged unconsciousness is rarely to be accounted for by a surface clot, although this possibility should always be eliminated by thorough exploration through suitably placed burr or trephine holes. Brain swelling presumably is not an important causative factor, as rigorous dehydration only occasionally produces improvement.

In many cases I believe that essential parts of the brain have been organically and permanently damaged by an intrinsic or diffuse neuronal type of injury, death being delayed only until failure of the general metabolic processes of what is virtually a brain-stem preparation.

In this section, however, I am principally concerned with patients showing prolonged mental symptoms, but on a different level. In this group of cases the patient recovers satisfactorily up to the stage of mild confusion, but then remains in a state of considerable mental impairment. For many weeks he may behave like a mental defective troubled with delusions.

For example, one of my patients would get up and walk about the ward in an aimless and purposeless way. He talked nonsense, and at night would barricade himself with pillows to protect

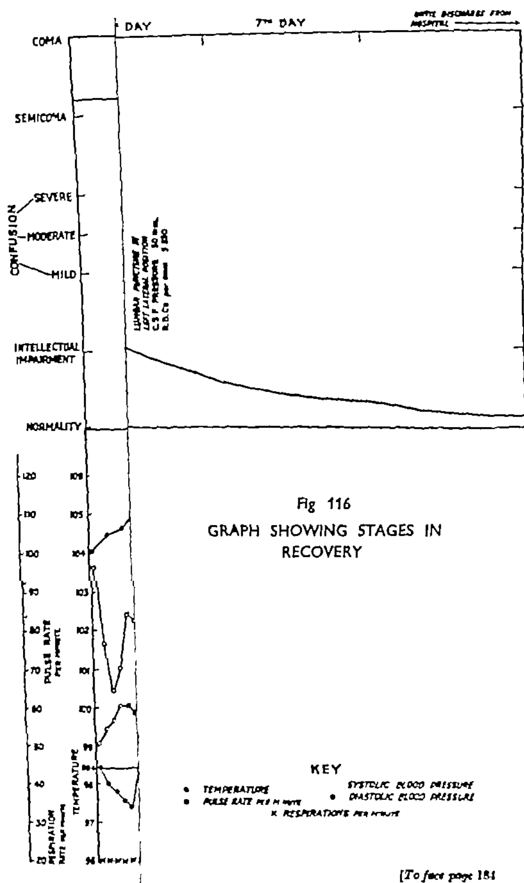
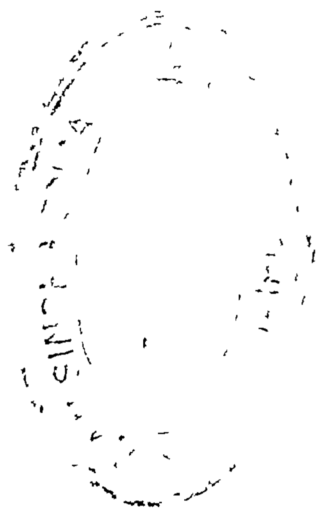


Fig 116
GRAPH SHOWING STAGES IN RECOVERY

[To face page 184



himself from the machine-gun fire from the man in the next bed. Another patient would persistently set off to meet some unknown person with whom he had no assignation. Another would get out of bed in the middle of the night and crow like a cock and mimic other animals to the annoyance of the other patients.

Such states most commonly occur in chronic alcoholics and those of poor intellect when the margin between normality and high mental defectiveness is a narrow one. In chronic alcoholics the state is essentially that of Korsakof's syndrome, confabulation being a common feature.

Brain swelling is occasionally the cause of prolonged intellectual impairment. Usually, however, the state seems to be due to an encephalopathy which is not progressive, as finally most patients make a satisfactory recovery.

The following is an important case, as it is typical of a group characterised by prolonged mental impairment resulting from or perpetuated by surface compression through collections of fluid in the subdural space.

On 28th October 1941 a man aged sixty four years fell down stairs and injured his head. When seen by his doctor about ten minutes later the man was severely confused. He rapidly became more deeply unconscious, and by the time he was admitted to hospital he was in an unconscious state bordering on semi coma. In three days time he had improved and was apparently out of danger.

On 13th November 1941, that is to say, sixteen days after the injury, he was admitted to the neurosurgical unit. He lay quietly on his back apparently in a peaceful reverie. There was normal tone in the facial muscles and occasionally he would focus his eyes on some near object which gave his expression a look of intelligence. He slept for long periods in the day and was quiet at night. He never asked for food but ate well when fed. He was incontinent of both urine and fæces if not regularly put on to a bed pan. By shouting he could be roused sufficiently to obey simple commands but his attention and concentration were poor. He was vaguely aware that he had received an injury but was completely disoriented as to time and place. When undisturbed he would lie motionless. He was never heard to speak spontaneously but when encouraged to do so would utter simple words or mutter incomprehensibly. His state was that of akinetic mutism.[1] There were no other abnormal neurological signs. There was no evidence of raised intracranial pressure: the optic discs were flat and the cerebrospinal fluid pressure 110 mm. as

[1] Cairns, H., Oldfield, R. C., Pennybacker, J. B. and Whitteridge, D. "Akinetic Mutism with an Epidermoid Cyst of the Third Ventricle." *Brain* 1941, 64, 273.

measured by manometry. In the fluid withdrawn there was 50 mg. of protein per cent., which is a little higher than normal. Radiography did not reveal a fracture of the skull.

On 20th November 1941, as there was no change in the man's mental state, an encephalogram was performed. The air studies showed that the anterior horns of the lateral ventricles were dilated and that a considerable quantity of air had collected over the frontal lobes in the subarachnoid spaces. The ventricular system was not displaced nor locally deformed. There was no air in the subdural space. The cerebrospinal fluid pressure was 100 mm. On this evidence it was thought that the frontal lobes of the brain had undergone atrophic change as the result of post-traumatic thrombosis. The Wassermann reaction in the blood and cerebrospinal fluid was negative. Previous to the accident the man had been mentally sound.

On 28th November 1941 there was again no appreciable change either mentally or physically. To confirm the diagnosis it was decided to explore the subdural space bilaterally and anteriorly. With the man in a supine position a trephine disc was removed on the right side of the skull over the frontal lobe, centred 2 in. from the middle line of the vault and just in front of the fronto-parietal suture. On incising the dura mater nothing abnormal was found in the immediate underlying subdural space On passing a curved dissector forward a cyst was opened and yellowish fluid welled up into the wound. A rubber catheter was passed into the cyst and 15 c c of fluid removed and collected. As this appeared to empty the cyst, the trephine disc was replaced and the skin incision closed in two layers. A similar exploration was made on the left side and a similar collection of fluid discovered and removed Immediately after the operation a lateral shoot X-ray in the nose-up position was taken and this revealed a large collection of air in the subdural space. The fluid from the cyst contained 800 mm. protein per cent —part of the fluid clotted.

The next day the man's mental condition had improved, and from this time onwards progress was slow but steady. He first began to take interest in his surroundings and sat up and began to ask questions. Later he fed himself and asked to go to the lavatory On 8th December 1941 he was still disoriented regarding time and place; he realised he had had an injury but did not appreciate its full implications. He had got over his akinetic-mutism but was still confused. He was allowed to return home on 8th December 1941. A month later the man was mentally normal. When seen in the Out-Patient Department two months later he was alert and cheerful and in every way a normal person. He did not remember anything about the accident or his stay in

hospital Six months after the receipt of his injury he was back at work carrying out his normal duties

McConnell[1] recently has written an important and interesting paper on this subject.[2] He has produced strong evidence that *local explorations are indicated in all cases of prolonged traumatic amnesia* He has shown that in these cases collections of fluid other than blood are occasionally found in the subdural space, and that when these are drained rapid and sustained improvement of the patient's mental state follows

POST-TRAUMATIC AMNESIA IN SIX CASES *

Case	Inaccessible Period.	Amnesic Confusion	Duration of Post traumatic Amnesia	
			Before Operation	After Operation
1		Severe	66 days	4 days
2		Moderate	11	4 "
3			21 ,	7 " †
4	3 days	Severe	9	6
5			14	6 months
6	4½ hours		6 "	21 days

* McConnell's Tables. † Died of pneumonia.

CHARACTER OF SUBDURAL FLUID *

Case	Side	Colour	Quantity	Protein (g /100 c c)	
				Subdural.	Lumbar
1	R	Slightly blood stained	Copious		
2	R	Clear	Few c c		
	L	Yellow brown	30 c c	3·5	0·05
3	R	None			
	L.	Slightly turbid	Copious		0·02
4	R	Yellow	Copious	2·0	0·03
	L.				
6	R	Blood-stained	93 c c		
	L.	Yellow	200 c c	1·8	0·03

* McConnell's Tables.

[1] McConnell, A A "Prolonged Post-traumatic Amnesia: Findings at Operation." *Lancet* 26th February 1911 273.

[2] Hawkes, C. D "Prognosis in Prolonged Unconsciousness following Head Injuries." *Amer Jour Surg.*, 1913, 62, 336.

STATUS EPILEPTICUS

Status epilepticus in infants and young children following a relatively mild injury to the head provides one of the most difficult diagnostic problems one is likely to meet in the study of cerebral trauma.

The following is a description of a typical case At ten o'clock one morning a child aged two years fell from his high chair in the presence of his mother. When picked up from the floor he was already unconscious and his face was of a deathly pallor. Indeed, the mother at first thought her child was dead Soon he began to stiffen and then to twitch all over his body The general practitioner was summoned, and he arrived one and a half hours later. At that time the child was in a condition of semicoma · also, it was judged that the right arm and leg were paralysed, the paralysis being of the flaccid type. From 11 30 A M. to 12 30 P.M the neurological picture was constantly changing: sometimes the tendon reflexes were active and sometimes absent, at times the pupils were small, at others dilated or unequal. There was a bruise on the left side of the scalp in the parietal area At 12.30 P.M the child was much more conscious and was beginning to open his eyes and to whine. Suddenly, and for no apparent reason, he went unconscious again and generalised twitchings developed

In view of the period of relative recovery followed by deepening unconsciousness, with the history of injury and with the sign of bruising of the scalp, a surface hæmorrhage of the extradural type was suspected. At this stage neurosurgical advice was sought; before the telephone conversation was concluded the convulsive seizure had ceased, and within half an hour the child had become less deeply unconscious. There was still, however, evidence of severe right-sided hemiplegia. By arrangement the child was transferred to the neurosurgical unit, a distance of forty miles, and arrived about 7 P.M.

On examination the child was found to be in a state of generalised continuous epilepsy. There was a small hæmatoma on the left side of the head, the pulse was running and the temperature was raised 1° F.

Diagnosis.—Correct diagnosis in this case was not only difficult but of fundamental importance in determining what should best be done to save the child's life.

The main possible pathological states that could account for the child's illness are as follows .—

(*a*) A large extradural or subdural hæmatoma, the epilepsy being a direct result of irritation of the brain by the clot.

(*b*) A large extradural or subdural hæmatoma, the epilepsy in this case resulting from bruising of the brain

(*c*) Epilepsy resulting from bruising of the cerebral cortex without compression of the brain by a surface blood clot.

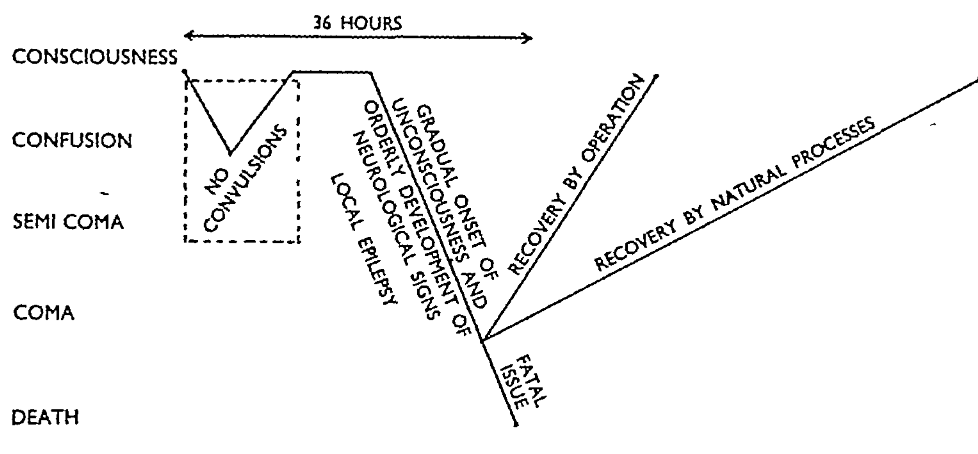
(*d*) Idiopathic epilepsy precipitated by injury, the injury having done only insignificant physical neuronal damage.

From the viewpoint of treatment the difference between groups (*a*) and (*b*) above is merely an academic one as is the difference between groups (*c*) and (*d*) Therefore the final decision that had to be made was whether a surface clot was present or whether we were dealing purely with a case of idiopathic epilepsy

Let us once again review the clinical picture as it developed from the moment of injury Whether the child fell as a result of loss of balance or because of an epileptic seizure cannot be known If the fact were known it would help little in the solution of the diagnostic problem, because an extradural hæmorrhage could as well result from the trauma of an associated fall as from the collapse of an epileptic seizure In this case the mother was sure the fall was accidental moreover, the child had not suffered previously from epileptic seizures and this information must always be sought so that an opinion can be given regarding the possibility of the recurrence of the seizures, should the child recover from the acute illness. Immediately after the fall the child was deeply unconscious Later, consciousness began to return There was evidence of damage to the head as shown by bruising of the scalp, and of actual brain damage as shown by hemiparesis. Later the child became once again more deeply unconscious.

The significance of these happenings, combined with the neurological signs, strongly pointed to a surface hæmorrhage From this point onwards, however, the march of events began to throw doubt on the diagnosis of a middle meningeal hæmorrhage For a second time the child began to regain consciousness, and such secondary recovery is a very rare occurrence indeed in the case of middle meningeal hæmorrhages. Also in most middle meningeal hæmorrhages the secondary deepening of unconsciousness is usually so gradual that the various mental and neurological changes are clearly observable In this case the second deterioration was sudden and was soon followed by violent twitchings all over the body In middle meningeal hæmorrhages muscular twitchings are rarely of the generalised convulsive type They occur usually as single or interval twitches confined to one limb or to one side of the body in other words, they are more

of the Jacksonian type. Also, nine hours after injury, in spite of prolonged and severe generalised twitchings, the child's condition did not look hopeless. Breathing was not stertorous, the pulse was racing but was full, the face was not expressionless and the jaw and tongue had not fallen to obstruct respiration. Finally, in a child as young as the one under consideration the chances



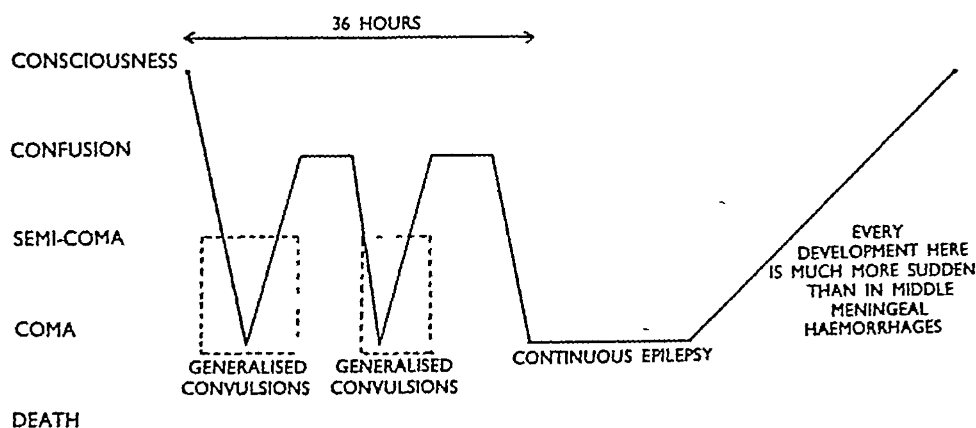


Fig 117

of epilepsy are far greater than those of a middle meningeal hæmorrhage.

Treatment—A diagnosis in this case was made of idiopathic epilepsy. Therefore, first of all 2 gr. luminal were given intramuscularly and then the fits were controlled by a few whiffs of chloroform applied by means of a piece of lint on an open mask. After the fits had been controlled the child soon dropped off into what looked like a deep and peaceful sleep. Had the child

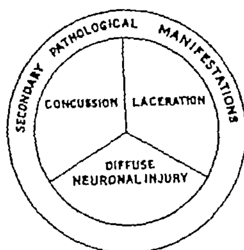


FIG 118

When the intrinsic injury is overwhelmingly severe the secondary pathological manifestations are not important

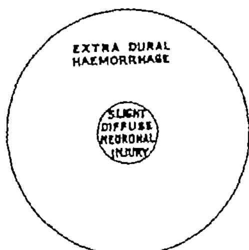
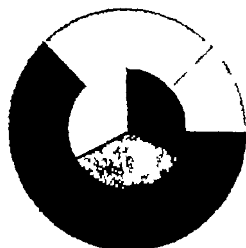


FIG 119

In favourable cases of extradural haemorrhage the intrinsic injury is minimal.



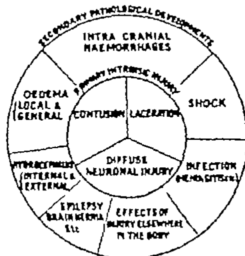


FIG 120

When an intrinsic injury is severe but not necessarily fatal the secondary pathological lesion can turn the scale adversely The meaning of the coloured circle is explained in the text

CHAPTER IV

TREATMENT OF CLOSED INJURIES OF THE HEAD AND SURGICAL TECHNIQUE

FIRST AID —First aid treatment on the roads is rarely the concern of medical men, since the injured rapidly come under the care of ambulance services and can, under normal circumstances, be removed to hospital within one hour When a decision regarding transport has to be made on those occasions when a tibia or femur has been broken, or when there is the slightest suspicion of a fractured spine, it is wiser to make the injured person comfortable and warm by covering him with coats and to wait for an ambulance rather than to send him to hospital huddled in the back seat of a private motor car

TRANSPORT

Patients with closed head injuries travel well in modern ambulances and may safely be sent long distances for treatment If distance, therefore, is the only consideration it is much wiser to transfer a patient directly to a hospital which is specially equipped for treatment of cerebral trauma than to an institution which is not fully conversant with this work

Many doctors are not willing to take the responsibility of transferring a patient with concussion from one hospital to another even though they wish to do so This difficulty can be readily overcome by telephoning a member of the special centre staff who will be only too willing to give advice The onus can then be placed on the special centre as to whether the patient should be transferred or visited

During transport a free airway must be maintained to prevent congestion and for this reason a comatose patient should be turned on to his side and not allowed to lie on his back in order to prevent his tongue from falling backwards to impede respiration (Fig 121) This position also allows saliva to trickle outwards which otherwise might be aspirated into the lungs A small dose

of morphia ($\frac{1}{4}$ gr.) may be given when a patient cannot be controlled by gentle restraint, but drugs are better withheld.

It is essential that the surgeon who is finally to take charge of the case should receive sufficient clinical data of the patient's early neurological state to enable him to proceed with treatment in a scientific manner. He must know, for example, whether



FIG 121

A patient in coma is best nursed on his side

the patient has been conscious at any time after the accident and, if not, how deeply unconscious he was when first seen. Also, if neurological observations have been made, and this is always desirable when conditions permit, as many details as possible should be passed on and a record of all drugs given.

NURSING

The surgeon in charge of a unit concerned in the treatment of cerebral trauma should personally instruct his staff in the details of nursing, and this responsible task should not be delegated to some impersonal lecturer. His Ward Sister should be made responsible for the recording of pulse rate, respiration rate and rhythm, temperature, amount of fluids taken and, under special conditions, for blood-pressure readings and changes in depth of unconsciousness. Records are best kept in the form of a graph on the same chart and preferably in different inks, so as to make them readily distinguishable and easily comparable. Observations in the first twenty-four hours should be made at half-hourly intervals and more frequently when active surgical measures are considered imminent. When a patient is obviously improving, hourly records are sufficient. These are the minimum requirements and are possible in the busiest wards. As the staff becomes more highly trained, details, particularly about the state of unconsciousness, will become elaborated. It is by these means only that a continuous clinical picture can be

obtained, and this is necessary if a patient is to receive the best possible treatment. Time spent in the training of the nursing staff will be amply rewarded

Restlessness.—As soon as a patient reaches the restless phase his mind is open to suggestion, when a kind or encouraging word will often be of the greatest help, although at the time it may appear useless Therefore, before anything is done for a restless patient he should be reassured that it is for his good At times it is surprising how far he can be influenced by the spoken word

As restlessness is always a trying problem to nurses and attendants without special neurological experience, it is advantageous to stimulate their interest and to gain their co-operation by explaining to them the nature of the underlying disturbance If they realise that a subarachnoid hæmorrhage is a form of meningitis they will handle a patient gently and refrain from making any forcible movement of the limbs or neck which is likely to cause further pain

Since restlessness is, to some extent, the result of overaction of the subcortical centres as these are released from the restraining influences of the higher centres, it is wrong to attempt to control movements by forcible restraint as this will merely act as a further stimulus and the patient will continue to resist until he has exhausted himself By raising and padding the bed sides and by having someone constantly in attendance to prevent a patient from getting out of bed, he may be allowed considerable freedom of movement without injuring himself Moreover, there is no particular therapeutic merit in the old established habit of keeping the head low In fact there are disadvantages, particularly if stretching of the neck muscles causes discomfort and thereby increases restlessness Drugs in the early stages should be given sparingly, and particularly when prognosis is in the balance Also, it is safer to repeat small amounts than to give a stiff initial dose

A routine which I have found useful and safe is as follows First, soluble luminal (3 gr) is given intravenously, followed by chloral hydrate (10 gr) and sodium bromide (10 gr) by mouth When a patient will not swallow double doses of chloral and bromide are given per rectum The chloral and bromide may be repeated in two hours and afterwards at lengthening intervals until reasonable rest is procured Paraldehyde is also an excellent drug and 4 drachms per rectum may be given instead of the chloral and bromide Morphia is rarely needed, but $\frac{1}{4}$ gr in combination with luminal will often give a complete night's rest The principle to be observed in the administration of drugs is to give the minimal amount which will give the desired effect To give more is dangerous and to give less is useless Constant

medical supervision of drug administration is therefore essential, and should never be allocated to the discretion of a junior member of the staff.

Feeding.—Patients are usually able and willing to swallow. When they cannot do so they must be fed through a tube passed through the nose into the stomach. This tube is fixed to the cheek by adhesive plaster.

Glucose in water is the only drink which need be given in the first twenty-four hours, and the quantity is regulated by the amount of dehydration considered necessary to avoid cerebral œdema. It is obviously illogical to withdraw cerebrospinal fluid by spinal drainage or by intravenous therapy if unlimited quantities of water are simultaneously given by mouth Theoretically, when dehydration is considered necessary, fluids should be entirely withheld, but under these conditions a patient rapidly becomes toxic, his mouth becomes dried, his tongue furred and he soon looks worse than before treatment started. Therefore in an attempt to relieve possible swelling of the brain the general bodily needs must not be overlooked, and 1-oz drinks every hour are not only beneficial but also essential. This is the minimum quantity of fluid that should be given in the first twenty-four hours. On the second day larger quantities of fluid may be given according to the state of the patient. When unconsciousness is deep and cerebrospinal fluid pressure high, it should be limited to 2 pints Less should never be given. When consciousness is obviously returning, 3 pints may be given if the patient will swallow them. Milk drinks should be given alternately with glucose water.

On the third day, it is essential to introduce some kind of protein-containing food, otherwise a patient will start to live on his own tissues and will die from exhaustion Junket, egg custard, milk puddings and meat jellies will be found useful for this purpose. Fixed rules for feeding, of course, cannot be laid down, and diet must be adapted to the individual needs of each case. More fluid, for example, will have to be given to a patient who is perspiring or passing water freely than to one with dry skin and inactive kidneys To supply the bodily needs, about 5 oz. of milk or its equivalent is necessary every two hours during the day and every four hours during the night.

The Bowels.—There is a serious risk of incontinence in giving aperients to an unconscious or semiconscious patient, and obviously this is a very distressing condition when associated with restlessness. Whenever it does occur it should be corrected at once by the administration of a mixture containing bismuth and opium Magnesium sulphate by mouth is often advocated in

amounts large enough to produce a watery stool, but it is better, in my opinion, to empty the bowel by enemata and to use other methods for dehydration Cathartics such as calomel should never be given At best they cause discomfort and often produce colicky pains with resulting restlessness

The Bladder —Incontinence is not uncommon and necessitates constant changing of the bed linen if a patient is not to become uncomfortable or his skin broken. Retention is usually due to apraxia, that is, a patient is unable to empty his bladder because he does not know what to do In these cases catheterisation is necessary, otherwise the discomfort of a distended bladder will make him restless or compel him to get out of bed in an effort to relieve himself When a patient is approaching consciousness a bottle or a bed pan, according to sex, should be placed in a suitable position and the patient requested to pass water When this device proves successful it should be repeated at three-hourly intervals. To keep incontinent patients dry the following methods may be found to be useful In the case of a female a bidet is fitted and in that of a male a length of colostomy tubing is fixed over the penis with strapping, the urine collecting in a receptacle placed at the side of the bed

The Skin, Mouth and Eyes —The skin needs early and regular care otherwise it will soon be broken For this reason, as well as for convenience of examination, it is important to strip a patient immediately and not to let him lie in his clothes until prognosis becomes obvious. A difficult problem arises in a violently restless patient. If abrasions are to be avoided, the knees, elbows and heels must often be bandaged in wool pads The mouth should be kept clean to avoid parotitis and this is best done by winding a wisp of wool around a stout pencil of wood and by swabbing the inside of the cheeks, the teeth and tongue with a mixture of glycerine and borax water Metal forceps must not be used because the patient may clench his jaws and break his teeth. Also, the finger should not be used, as this may be severely bitten

When eyelids are swollen the conjunctival sacs should be irrigated regularly with boracic lotion

Temperature —It is usual to place a shocked patient in a radiant heat cradle With proper precautions this is a wise measure, but a warning must be given since the thermo-regulating centres are often temporarily in abeyance and the body tends to take on the temperature of its environment. An excessive amount of heat may therefore be applied, which not only causes an undesirable loss of fluid by sweating but also may lead to hyperthermia. Such complications may be avoided if the patient's temperature is taken at regular intervals

A rise in temperature of 2° F. or more should be treated by tepid sponging, or by cold packs and intravenous injections of aspirin (20 gr.) if the temperature cannot be controlled by other means.

Absorption of blood in subarachnoid hæmorrhages often causes a rise in temperature of 1° to 2° F.

Hypostatic pneumonia may occur, with or without obvious signs referable to the chest, when a rise in temperature may be the only sign of its onset. A rigor must be reported by the nursing staff at once, as this often indicates the onset of meningitis. Immediate chemotherapy in such cases is necessary if the patient's life is to be saved. Severe intrinsic injuries, particularly of the hypothalamus, commonly lead to death in hyperthermia, for which no kind of treatment is of any avail.

SPECIAL FORMS OF TREATMENT

Lumbar Puncture and Manometry.—In most cases, despite restlessness, a lumbar puncture can be done satisfactorily under local anæsthesia. A general anæsthetic must never be given to quiet a restless patient, as this will not only give a false pressure reading but is, in itself, dangerous. Usually two assistants are required to bring the patient into the necessary position and to hold him still while the puncture is made (Fig. 122) The patient is brought to the edge of his mattress under which a bed board has been placed, and his hip joints are fully flexed and his head bent forwards. A sterile towel is pushed beneath the patient's back by the operator and full aseptic precautions are taken By means of a hypodermic needle the skin between the third and fourth lumbar spines is anæsthetised and the manometric needle is introduced exactly in the midline and angled backwards about 10°, so that it will travel a little towards the head The resistance of the interspinous ligaments will be encountered and a jerk will be felt when the needle pierces the ligamentum subflavum and the dural envelope. Absence of resistance means that the needle has left the middle line and may slide beside the theca and spike a posterior root When this happens, or when a bony resistance is met, the needle must be immediately withdrawn and a fresh attempt made. A common mistake is to introduce the needle too deeply so that it passes through the far wall of the dural sac. This mistake can be avoided by withdrawing the stylet frequently to see if cerebrospinal fluid has been tapped and by rotating the needle so as to remove its bevel from the possible obstruction of a posterior root which may have floated against it. A dry tap indicates faulty technique.

When intracranial pressure is high, withdrawal of cerebrospinal

fluid may lead to a tentorial or cerebellar pressure cone with resulting death. The danger of this complication, however, is outweighed by the importance of the information that only manometry can give.

As stated previously, pressure is measured in millimetres of cerebrospinal fluid, the normal range being between 50 and 150 mm in the horizontal position

The First Twelve Hours—Primary shock is treated by warmth and rest and in the majority of cases no other treatment is needed Apart from complications elsewhere in the body, loss of

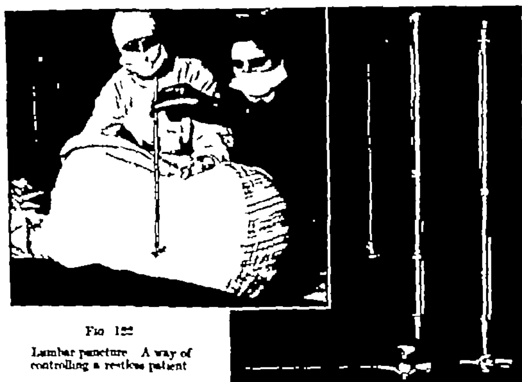


FIG 122

Lumbar puncture A way of controlling a restless patient

blood in the immediate stages of a closed head injury is usually so small that blood transfusions are rarely necessary In those cases when the blood pressure remains persistently low (100 mm. Hg and below) a small amount of blood (250 c.c.) may be given with advantage and may have to be reinforced by suitable stimulating drugs. These however, are usually better avoided

Urgent associated injuries, such as rupture of abdominal viscera must be treated irrespective of the degree of concussion, for the obvious reason that the patient will die if this is not done Fractures should be splinted in the first few hours to prevent further deformity but on no account should they be forcibly manipulated as this seriously aggravates shock Excision of compound fractures may safely be left for eighteen hours if the

condition of the patient is such as to necessitate the delay, and particularly if the operation cannot be done under local anæsthesia It cannot be too strongly stressed that general and even spinal anæsthesia is extremely dangerous in the early stages of concussion, since any further change in intracranial tension or blood pressure may render inadequate a cerebral circulation which is already embarrassed by pathological changes within the skull.

After Twelve Hours.—A lumbar puncture is performed and a measurement taken of the cerebrospinal fluid pressure When the pressure is below 150 mm nothing further need be done, as this lies within normal limits. When it is above 150 mm., fluid should be withdrawn until the pressure is reduced to 50 mm This figure was chosen because it is the minimal normal and therefore allows of the maximal accumulation of cerebrospinal fluid before an abnormally high pressure is reached again. When the cerebrospinal fluid is heavily stained with blood it should be freely withdrawn at four-hourly intervals until active bleeding, as shown by cell counts, has ceased.

After Eighteen Hours—Whether a patient is improving or not a lumbar puncture should be performed at the end of eighteen hours and sufficient fluid withdrawn to bring the pressure to 50 mm Should a pressure above 200 mm be found at this second reading, spinal drainage will have to be reinforced by some other kind of dehydration, and the choice of the method will depend on the condition of the patient's circulation. If the pulse is full and bounding, 3 oz of magnesium sulphate dissolved in 6 oz. of water should be introduced slowly into the rectum by means of a funnel and tube and great care taken to see that it is retained. When the circulation is feeble or normal, 75 c.c. of 50 per cent. solution of sucrose should be injected intravenously. A profuse subarachnoid hæmorrhage, however, is a contraindication to intravenous therapy, since further bleeding may be aroused if the blood pressure is raised.

The general metabolic processes must also be considered as well as the local conditions inside the cranium Dehydration, therefore, must not be carried to the extent of damaging the other tissues of the body in an attempt to overcome hypothetical cerebral œdema or excessive secretion of cerebrospinal fluid Hypertonic salines are also contraindicated in old people whose tissues are already dehydrated.

After Twenty-four Hours—The cerebrospinal fluid pressure is checked every twelve hours until the patient is conscious, and kept within normal limits, by lumbar drainage, reinforced if necessary by dehydration. Fluids are given in sufficient quanti-

ties to maintain the circulation and blood transfusion also will be necessary should the patient's pulse begin to deteriorate

Dehydration—Dehydration is an attempt to reduce or control excessive formation of tissue fluids other than blood in the brain These fluids usually accumulate in the interstitial spaces and, more rarely, within the cells themselves The object of dehydration is to prevent the development of cerebral œdema and so regulate intracranial pressure that —

(a) The optimum conditions for the recovery of damaged nerve cells are produced,
(b) Cerebral congestion is avoided, thereby preventing faulty cerebral circulation with resulting faulty tissue metabolism,
(c) Dangerous herniations do not develop which may compress essential parts of the brain

Dehydration may be carried out in two ways, either through the intestinal canal or by intravenous therapy

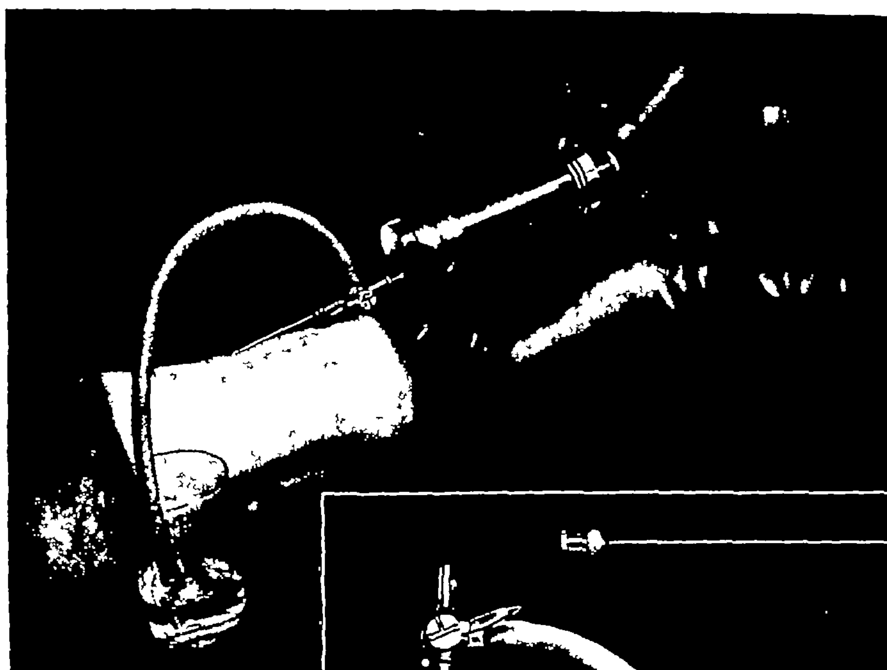
Intestinal Dehydration.—(i) Oral administration of magnesium sulphate the patient is given magnesium sulphate by mouth until a watery stool is produced and this condition maintained by further dosages of the drug The difficulty in this form of treatment is to prevent excessive purging In any case, the magnesium sulphate is apt to cause intestinal discomfort with resulting restlessness Moreover, the patients are incontinent, and this leads to a very serious nursing problem Magnesium sulphate by mouth is an excellent remedy for relieving symptoms in a patient who has regained consciousness but who is complaining of headaches or dizziness, or who shows mild mental torpor or deterioration due to brain swelling

(ii) Magnesium sulphate enemas 3 oz magnesium sulphate dissolved in 6 oz water are slowly introduced into the rectum by means of a funnel and tube, great care being taken to ensure that the fluid is retained The enemas are repeated at eight or twelve-hourly intervals until the patient is conscious or until the rectum becomes intolerant of further injections

Intravenous Dehydration.—For intravenous transfusions of hypertonic solutions the apparatus is assembled as shown in Fig 123

The patient's arm is held by an assistant either along the edge of the bed or in abduction, according to the position which is most convenient to the surgeon and the veins are made to distend by blowing up the arm band of the sphygmomanometer to venous pressure After the syringe has been partly filled with a 50 per cent solution of sucrose through the side tube the needle

is pushed into the vein, care being taken not to pierce its distal coat. The band of the sphygmomanometer is now allowed to



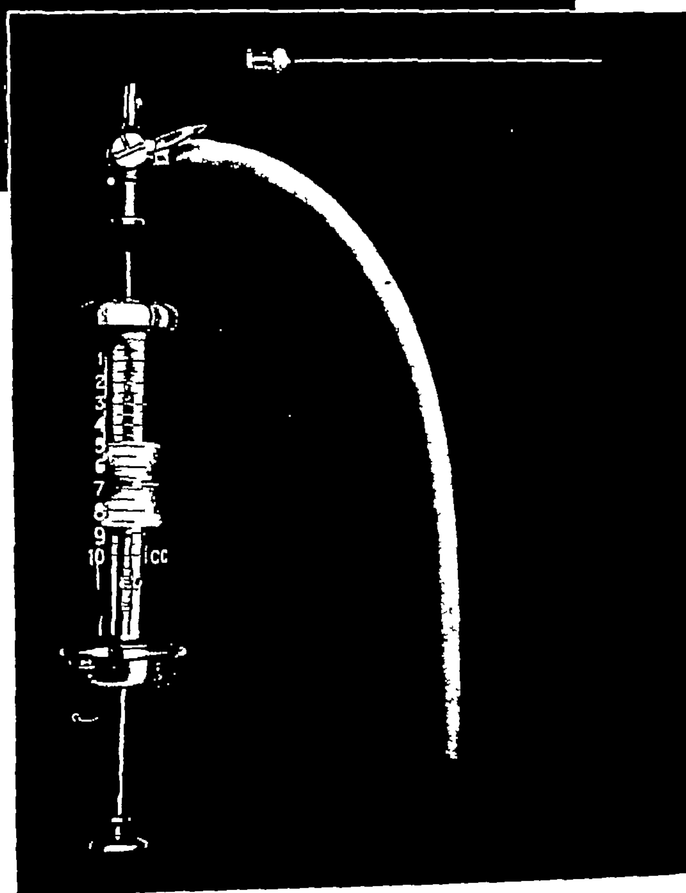


Fig 123

An intravenous transfusion.

deflate. By means of the stop-cock the syringe can be refilled without disturbing the needle and the injection continued until the required amount of fluid has been given. Care must be taken to keep the needle within the lumen of the vein, as leakage of

hypertonic solutions in the subcutaneous tissues will cause a painful arm or may even lead to sloughing of the skin If the tissues swell as the injection is being made, this means that the needle is not inside the lumen of the vein When extravasation does occur the sucrose solution may be diluted by injecting sterile water into it, alternatively, the extravasated fluid may be let out through a small incision in the skin

Sucrose, glucose and sodium chloride are most commonly used for hypertonic solutions, sucrose and glucose are used in 50 per cent solutions and as much as 100 c.c. may be given at one injection, sodium chloride is used in 15 per cent. solutions and the maximum amount that may be given at a single injection is 35 c.c The advantage of sucrose over the other two substances is that its dehydrating action is more prolonged and that it is much less apt to cause secondary waves of cerebral œdema.

According to Temple Fay[1] the treatment of acute cerebral trauma by means of dehydration originated in 1921 in the neurosurgical service of Dr Charles H Frazier of Philadelphia. Certainly it was due to the vigorous advocacy and writings of Fay that for a time dehydration therapy became almost a routine in the treatment of acute injuries of the head The pendulum has now swung in the opposite direction although dehydration still has its strong supporters For example Professor Lambert Rogers[2] recently has brought forth evidence to show that rectal dehydration has considerably improved his results in a large series of head injuries

The objections to intravenous dehydration are as follows —

1 There is no proof that cerebral œdema is an important factor in the production of symptoms in the early stages of trauma.
2 It may increase bleeding
3 It may interfere seriously with the general metabolic processes of the body
4 The tissues of the body may be over-dehydrated with resulting toxæmia.
5 Lumbar puncture may be impossible in a restless patient, and in these cases it is wrong to dehydrate on the assumption that the pressure is raised
6 Theoretically it is inconsistent to dehydrate and to give fluids by mouth

[1] Fay T "The Treatment of Acute and Chronic Cases of Cerebral Trauma by Methods of Dehydration. *Ann Surg.*, 1935 101, 76.
[2] Rogers, L. "The Treatment of Cerebral Contusion. *Brit Med Jour.*, 1913, 1, 151

My own opinions are :—

(*a*) That dehydration is not indicated in the acute stages of cerebral trauma save in those cases where intracranial pressure is raised and where this rise of pressure has been proved by exploration to be due to œdema of the brain.

(*b*) That dehydration is necessary if, after eighteen or twenty-four hours, the cerebrospinal fluid pressure remains persistently high in spite of lumbar drainage.

(*c*) That dehydration is indicated in cases of prolonged unconsciousness when doubt as to the presence of a surface clot has been eliminated

(*d*) That dehydration is indicated in the stage of convalescence when there is persistent intellectual impairment, mental torpor or headaches, etc.

Hypotonic Therapy.—Occasionally following an acute head injury the brain shrinks and the intracranial pressure becomes negative instead of positive. This happening may be suspected when the manometric pressure, as measured by lumbar puncture, is below normal It can be proved by means of inspection holes. These rare cases of brain shrinkage are best treated by intravenous injections of distilled water, 50 c c. at first being given and this dosage repeated at half-hourly intervals according to the effect on the patient. Shrinkage of the brain and negative pressures often follow hypertension and swelling of the brain, and merely indicate that the cerebral circulation is failing and that death is impending ; indeed, the tension conditions found at post-mortem are very different from those which obtain during that period when the patient is fighting for his life and prognosis is in the balance.

In the treatment of chronic subdural hæmatomata after the subdural blood has been evacuated and when the brain tissue refuses to expand to occupy the subdural space, intravenous injections of hypotonic distilled water should be given until the desired conditions of brain swelling are obtained Voris[1] also advised the following manœuvre to encourage the brain to return to its normal volume and shape The subdural space is filled with distilled water and a soft drainage tube is led to the exterior and clamped. The wound is then reformed in layers firmly around the tube. Immediately after the patient has been returned to bed 1,000 c.c distilled water are given intravenously followed by 1,000 c.c. isotonic solution of sodium chloride. The clamp

[1] Voris, H C "Subdural Hematoma" *Jour Amer Med Ass*, 23rd November 1946, 132, 686 692

is removed from the rubber tube after six hours, and the tube itself withdrawn twenty four hours later

Chemotherapy.—In every case where a fracture involves the paranasal air sinuses a patient should immediately be put on a full course of chemotherapy to prevent the development of meningitis. The fracture may be demonstrated directly by radiography or inferred when there is a profuse and persistent discharge of blood from the nose with bruising and swelling about the forehead and eyelids. Cerebral rhinorrhœa, of course, is a certain indication of a fracture of the above type.

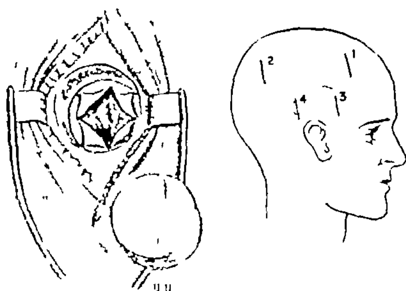


Fig. 124.
Inspection holes.

Inspection Holes (Fig. 124).—Local exploration through burr or trephine holes has solved the problem of deepening unconsciousness resulting from increasing surface hæmorrhage. In any case, when diagnosis is in doubt on purely clinical grounds a local exploration should always be made. Provided that the exploration is made under local anæsthesia and with reasonable neurosurgical precautions no harm is done to the patient, even if the finding do not lead to useful operative procedure.

Site of Operation and Surgical Procedure.—1 In cases of suspected extradural hæmorrhage the hole is placed according to the neurological evidence or to the presence of fracture lines. When made in the classical position about the pterion, the skin

incision should be made vertically, so that if a hæmatoma is found the incision can be curved upwards and backwards to fashion a formal subtemporal exploration. When made elsewhere, over a fracture or sprung suture line, the skin incision should be so fashioned that it will conveniently form the limb of a wider exploration (a flap) if this should prove necessary, as it often does when an extradural clot is in an atypical position (occipital).

2. When localising signs are present the hole in the skull is placed according to the neurological evidence; for example, in cases of hemiplegia it is made over the opposite motor cortex. When aphasia is present, two holes are placed over the speech centre, that is, low on the left side of the head in right-handed people—one in front of and one at the back of the temporal fossa.

Given that an extradural hæmorrhage is not found, the dura is incised in a cruciate manner, care being taken not to open the arachnoid mater, so as to give opportunity for examination of the subdural space. If the dural space is empty the arachnoid mater is incised Often at this point yellowish or blood-stained fluid spurts up into the wound This high intracranial pressure is often found when lumbar manometry has given a normal or near-normal pressure reading. A small drainage tube is passed down to the dural opening but not through it, and the wound reformed in two layers. Great care must be taken with subsequent dressings to prevent infection, as the cerebrospinal fluid spaces have been left open The tube is removed in twenty-four hours and the opening in the skin closed with a stitch. The above operation constitutes drainage of what is virtually a state of external hydrocephalus, and possibly it is one of the best and surest methods of maintaining intracranial pressure at that level which gives the optimum conditions for the recovery of brain damage

When, on opening the arachnoid mater, little subarachnoid bleeding is found and the brain bulges into the wound, then we are dealing with brain swelling due either to œdema or to intracerebral hæmorrhage.

3. In acute subdural hæmorrhages the blood tends to trickle to the most dependent parts of the subdural sac. Thus with the head in the upright position most of the blood will accumulate over and under the temporal lobes of the brain. As bilateral bleeding is common, bilateral exploration is always indicated, whether or not the hæmorrhage is found on the side on which it is first sought The exploration holes are placed low in the middle of the temporal fossa (see Fig 124). If on opening the dura mater a subdural hæmorrhage is tapped, then one of two procedures is possible:—

(a) The hæmorrhage is aspirated as completely as possible by means of a sucker with a curved end and the cavity drained with a tube for forty-eight hours No attempt is made to find the bleeding point, as it rarely lies beneath the local exposure Sealing of the ruptured vessel is left to natural processes

(b) A wide osteoplastic flap is turned, or a subtemporal decompression made, in order to facilitate more adequate removal of the extravasated blood which is often extensive, also the wide exposure permits of search for the bleeding vessel which, if found, can be sealed by one of the methods to be described later THIS RADICAL PROCEDURE IS RARELY INDICATED, AND IN MY OPINION SHOULD ONLY BE CARRIED OUT WHEN A DEFINITE LACERATION OF THE BRAIN IS SEEN THROUGH THE LOCAL EXPOSURE OR IN THE CASE OF A LARGE GELATINOUS CLOT As a general rule it can be laid down that in an acute head injury the simpler the operation the greater are the chances of recovery

4 In chronic subdural hæmatomata, when localising signs are present, a 1 in trephine hole is made over the presumed centre of the clot When localising signs are absent, holes are first bored over both parietal eminences and if a clot is not found here further holes are made just above the Sylvian points, as shown in Fig 124 Before abandoning the search for a chronic subdural clot, both sides of the skull must be explored at the four above-mentioned positions

The advantage of a trephine hole over a burr hole is that the disc of bone removed may be replaced and the skull thus repaired if drainage at a particular site is not found necessary This procedure, as will be shown later, will eliminate many difficult medico-legal arguments which otherwise might arise

Clots are easily recognised by their black dark blue or greenish yellow colour When they are present a cross-incision is made through the dura mater and the underlying adventitious membrane The fluid contents are evacuated by suction through a suitably curved nozzle All accessible adventitious membrane should be removed by dissection When the resulting cavity is large, a second trephine hole should be made at a distant periphery, and the remaining fluid syringed out by injecting saline into one hole and letting it escape through the other Finally, small drainage tubes are inserted and kept in position for forty eight hours or longer according to the continuation of the discharge (Fig 126)

Occasionally strap-like bands of thickened arachnoid deep

in the wound have to be divided through an osteoplastic flap in order to let the brain expand.

Convalescence may be rapid but is often slow, causing anxiety even in those cases that will finally do well. Should the patient's condition deteriorate at any time after operation, it is imperative to reopen the wound immediately to ascertain whether the clot has reformed or not Also, the opposite side should be explored because subdural hæmatomata are often bilateral. When a clot



Fig 125
The extent of the subdural hæmorrhage drained in this case is outlined by the air shadow The drainage tube is still *in situ*

is suspected but escapes detection by exploration, pneumencephalography always discloses its position and size.

In those cases when the adventitious membrane is very thick, or when the clot is organised or calcified, the membrane or clot must be removed by dissection through the wide exposure of an osteoplastic flap.

It is a great tragedy to let a patient die from a subdural hæmorrhage when his life can be saved by so simple an operation as that described above. In fact, when in doubt regarding diagnosis, the presence of a subdural hæmorrhage can be confirmed or eliminated by early local exploration.

5 In cases of prolonged unconsciousness an extradural or subdural hæmorrhage should be eliminated through burr holes made in the four positions used for the exploration of a chronic subdural hæmatoma.

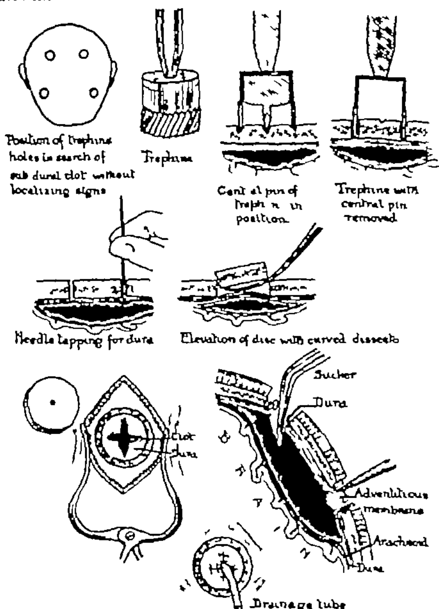


Fig. 126
Evacuation and drainage of a chronic subdural hæmatoma.

6 In cases of prolonged confusion or mental impairment burr holes are made over the frontal lobes (Fig 126)

Subtemporal Explorations and Decompressions—The term subtemporal decompression, by common usage has come to mean the removal of bone beneath the temporal muscle but does not necessarily imply that an opening is also made in the dura mater

To avoid ambiguity, the term "decompression" is here used to denote that the dura mater has been widely opened to relieve tension within the dural envelope; and "exploration," that the bone only has been removed for purposes of inspection of the extradural space. A decompression must always be made over a silent area, since the part of the brain uncovered is apt to herniate through the dural opening and to become strangulated or contused with consequent loss of function. A left-side subtemporal decompression is not entirely without danger, as an aphasia might ensue. Therefore whenever the clinical indications do not demand an exposure elsewhere, a decompression should always be made on the right side beneath the temporal muscle and over the temporal lobe of the brain.

SPECIAL INSTRUMENTS AND MATERIALS (Fig 127)

1 Safety-razor
Lintine and dental swabs
1 Toothed and 1 non-toothed dural dissecting forceps
1 Curved dissector (Adson type).
1 Rougine.
1 Teaspoon
1 Saline syringe (C Ryle)
2 Flat metal brain retractors
2 Self-retaining retractors
1 Michel clip apparatus
1 Hudson's brace (large handle pattern)
1 Perforator
1 Set of graded burrs
1 Trephine (author's pattern to fit brace)
1 Wilms forceps
1 Double-acting nibbling forceps
1 de Vilbiss forceps
2 Gigli saw handles
2 Gigli guides and saws
1 Sucker apparatus
1 Diathermy apparatus, leads and electrodes
1 Electromagnet
1 Overhead table or tray
1 Silver clip apparatus
6 Rubber bands
Horsley's wax
Waxed silk
12 No 15 Bonney's needles (half-circle)
1 Headlight
At least 3 dozen fine-pointed hæmostats

Preparation of the Scalp.—The hair is cut short with scissors or clippers and the whole head is shaved with a safety-razor, great care being taken not to break the skin. A skilled barber, of course, may use any method he wishes. After shaving, the scalp is washed and scrubbed with soap and water and then swabbed with perchloride lotion. Finally, it is treated with a solution of dettol and wrapped in a sterile compress Iodine or spirit should not be used as they are apt to scale or burn the skin. Careful preparation of the scalp is important if infection is to be eliminated, and complete shaving is also necessary, since it is impossible to know before operation whether both sides of the skull will have to be opened or not.

Anæsthesia.—*Operations for closed head injuries should be done whenever possible under local anæsthesia.* When, for reasons of rest-

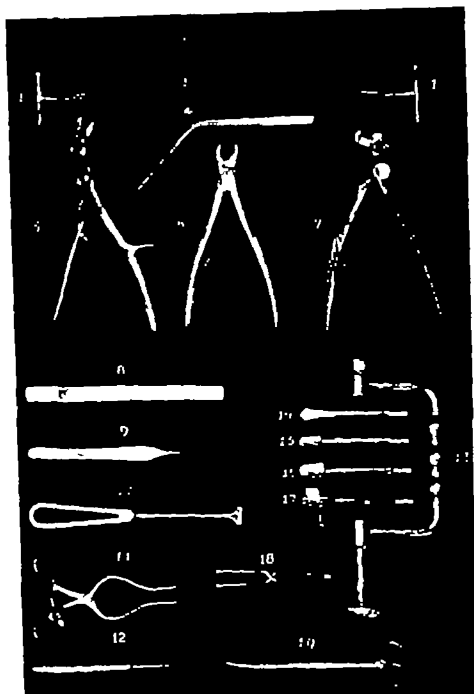


Fig 1—

Special instruments.

1 Gigli saw handle
2, Gigli guide
3, Gigli saw
4 Sucker end
5, Double acting nibblers.
6, Wilms forceps.
 d Allies forceps.
8 Brain retractor
9 Dural dissecting forceps.
10, Dural scissors.
10, Rake retractor
11 Self retaining retractor
12, Adson curved dissector
13, Hudson brace
14 Perforator
15, Burr
16, Burr
17 Trephine to fit brace
18 Michel clip applicator

lessness, a basal or general anæsthetic or a combination of the two has to be given, the chances of recovery are seriously diminished.

When a patient is sufficiently unconscious to tolerate intubation without general anæsthesia, an intratracheal tube lubricated with percaine ointment (10 per cent.) should be introduced and connected through a suitable apparatus with an oxygen cylinder This precaution will often save the lives of those patients whose respiratory centres fail before there has been time to relieve the compression of the brain. Care in these cases must be taken not to overdistend the lungs by blowing in oxygen too rapidly.

Restlessness under local anæsthesia may be due to (1) suffocation, (2) overheating under wet towels, (3) pain in the wound owing to inadequate anæsthesia and (4) postural discomfort Before resorting to drugs to quieten a patient each of these factors must be reviewed and corrected if necessary. When a patient continues to be restless for reasons other than those of faults in operative technique, an intravenous injection of Pentothal, Evipan or morphia is necessary. In my opinion, Pentothal in the hands of an expert anæsthetist is an excellent anæsthetic

The line of an incision is infiltrated with novocaine-adrenalin solution until the skin is raised into a distinct mound. Whether the anæsthetic is injected above or below the galea appears to make very little difference to the resulting anæsthesia, but as Dott has pointed out, the correct anatomical layer is superficial to the galea as it is there that the nerves are found (Fig. 128). The needle is then inserted at right angles to the bone and the pericranium injected at intervals of 1 in. Further injections are made according to the nerve supply of the part to be exposed, and these details will be given later when the various operations are described. A generous amount of anæsthetic should always be used; up to 100 c.c of 1 per cent. novocaine solution to which 5 drops of adrenalin (strength 1 in 1,000) have been added may be injected with safety. At least five minutes should be given to allow the novocaine to act before making the incision, because a painful start may rouse the patient and make him restless for the remainder of the operation. Further injections of anæsthetic are often necessary in the later stages of an operation, particularly when the wound is being closed. Local anæsthesia should not be omitted in a deeply unconscious patient because an unblocked incision will inflict further shock. Moreover, a patient is apt to become more conscious as a compression is relieved, and resentment of a painful unanæsthetised wound may be shown by uncontrollable restlessness.

If, during an exposure under local anæsthesia, a patient becomes so restless that operative procedure is no longer safe, he may readily be quietened by an intravenous transfusion of Pentothal.

Intravenous anæsthesia under these conditions is, in fact, the method of choice

If, before operation, a patient is uncontrollably restless and it is

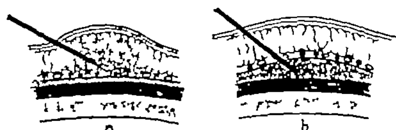


Fig 128

a Correct novocaine infiltration of scalp. The solution is diffused in fibro-fatty tissue and acts on larger nerves and vessels in its deepest layer *b* Incorrect infiltration: the needle has passed too deep and its point lies in subaponeurotic areolar tissue where the solution is ineffective on scalp as aponeurosis is relatively impervious. (*Norman Dott.*)

obviously impossible to operate on him under a local anæsthetic, some form of general anæsthesia is necessary Whatever type of anæsthesia is chosen whether intravenous or by inhalation,

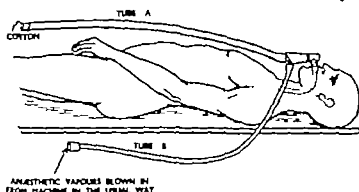


Fig 129

Ayre's tube

When a general anæsthetic has to be administered for the treatment of a head injury care must be taken to provide a free airway and to maintain full oxygenation. Congestion and straining lead to serious cerebral complications and make surgical manipulations difficult The tube A forms a reservoir and ensures an adequate concentration of anæsthetic vapours Moreover a *certain amount of rebreathing occurs which keeps the carbon* dioxide gas at a sufficiently high pressure to stimulate the medullary respiratory centres. No strain is thrown on the lungs, as occasionally happens when a rubber bag and valve are used. Rate of respiration can be seen by the movement of a piece of cotton which hangs by strapping over the opening of tube "A". Tube "A" must be at least [illegible] ft in length although it may be longer and must be as wide as the diameter of the trachea.

I believe intratracheal intubation is essential to maintain a satisfactory airway and adequate oxygenation If inhalation anæsthesia is chosen and this is my method of choice every effort must be made to prevent straining and congestion at

intubation as well as during operative procedure. Serious harm can be done at induction of anæsthesia if great care is not taken. To facilitate the introduction of a tube into the trachea an intravenous injection of Pentothal is useful. Whether trilene, ether or chloroform is used to continue the anæsthesia seems to make little difference provided the following conditions are observed —

(*a*) The patient is kept light.

(*b*) The airway and oxygenation are perfect

Pre-operative injections of morphia are contraindicated Atropine may be given When the medullary centres are embarrassed as the result of the injury, prolonged anæsthesia under Pentothal is dangerous. Gas and oxygen alone nearly always lead to straining and congestion even in the hands of skilled anæsthetists.

Basal anæsthetics such as Avertin, I believe, are dangerous and are better avoided. The administration of ether and chloroform by the open method cannot be too strongly condemned, because these anæsthetics increase intracranial pressure and venous congestion to a degree that makes intracranial manipulations impossible or exceedingly dangerous.

General Theatre Technique.—Correct posturing and towelling on the operating table are so important that they can make all the difference between a successful and unsuccessful operation.

The patient is placed supine or prone on the table according to the part of the skull which it is proposed to open. For example, in a right-sided subtemporal decompression he should lie on his back with the right shoulder raised and his face turned to the left side. He is strapped into position to prevent him from slipping off the table should he become unruly, and his wrists are loosely tethered to avoid the danger of his putting his hands into the wound. An easy way to secure the patient is to pass folded sheets over the chest and thighs, and after overlapping their ends under the table, to fix them in position with a line of safety-pins. The hands can be controlled by a bandage knotted into a clove-hitch and passed over the wrists and then attached to the sides of the table. The limbs should rest in a comfortable position and painful pressure points should be avoided by padding the table with pillows. The head is raised above the rest of the body to promote venous drainage and the operation field should be easily accessible to both the surgeon and his assistant (Fig. 130). A linen towel over a square of batiste is placed beneath the head, a large lint guard wrung out in perchloride lotion is folded over the scalp and four large towels wet with perchloride lotion are used to shut off the operation field. The ends of the towels towards the face are lifted from the forehead and draped across an overhead table

or tray so as to keep the patient's nose and mouth free This ensures a free airway Finally, a large sheet with an opening in one end is used to drape the whole field The surgeon and his assistant stand on each side of the patient s head, and the two theatre nurses are placed on raised stools at each end of the over head table (Figs 131 134) If the towels are clipped to the lint guard and the guard sewn to the anæsthetised edge of the wound, the towels will follow any movement of the patient s head should he become restless

The Operation of Subtemporal Exploration through a Muscle Split.—The skin incision is marked out with an iodine line It

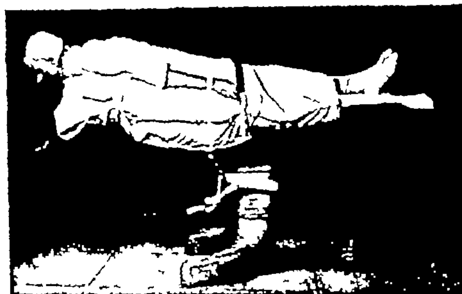


Fig. 130

A patient placed in position on the operating table for a subtemporal decompression under local anæsthesia.

starts at the upper border of the zygoma $1\frac{1}{2}$ in in front of the external auditory meatus and runs upwards for $2\frac{1}{4}$ in., then curves horizontally backwards for $\frac{3}{4}$ in The classical incision, consisting of the vertical limb only gives an inadequate exposure particularly as the temporal muscle is often thickened with blood and consequently so rigid that mobilisation is difficult.

Local anæsthetic is injected along the line of the incision and along its base from the margin of the orbit anteriorly to the mastoid bone posteriorly, particular care being taken thoroughly to anæsthetise the tissues above the ear The needle is then inserted at various points at right angles to the skin and the lower part of the temporal muscle is freely infiltrated down to the bone so as to anæsthetise the deep temporal nerves as they turn upwards If a generous amount of anæsthetic is used the whole of the temporal fossa can be made completely insensitive

Fig 131 —An overhead operating table (Author's design)



Fig 132

Position of the patient under the overhead table Note how the towels are draped away from the face, ensuring a free airway Note also the position of the anæsthetist

Fig 133
A method of towelling



Fig 134
An operating team in position.

Each side of the incision is then compressed by the finger-tips of the assistants to control bleeding and to pull the wound apart as the skin and deep fascia are incised. The galea is picked up with artery forceps at intervals of ½ in It is unnecessary to secure

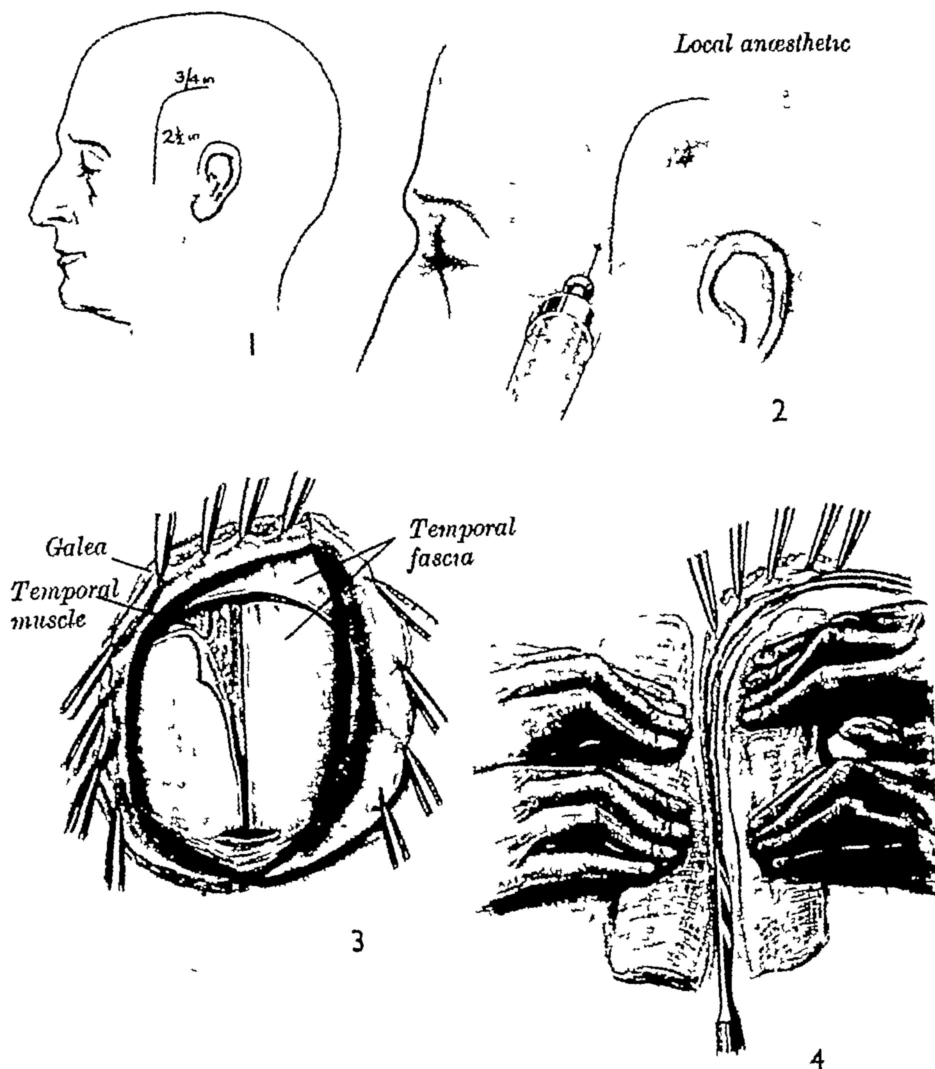


FIG 135

Subtemporal exploration by muscle split

1, Line of incision 2, Zone of anæsthesia
3, Method of reflecting the temporal fascia
4, Exsanguination of the skin by digital compression as an incision is made

each bleeding point separately, since the drag of the artery forceps will produce satisfactory hæmostasis when they are thrown over the edges of the wound. The triangular skin flap is reflected backwards and the skin at the anterior end of the wound is undermined. The artery forceps are arranged into neat bundles with

elastic bands and pinned to the towels so that they do not obstruct the operation field An alternative method of hæmostasis is to clamp tetra cloths into position with a series of closely placed Michel clips

By means of an incision shaped as shown in Fig 135 the temporal

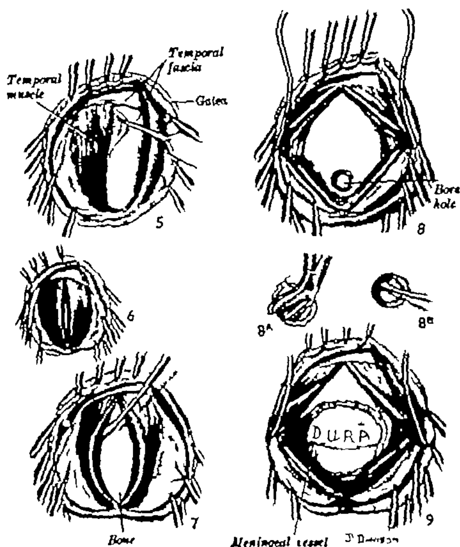


FIG. 136

5, Exposure of the temporal muscle
6, Splitting the temporal muscle
7 Separation of muscle from bone
8 Exposure of bone
8A, Removal of bone by nibbling.
8B, Separation of dura from bone
9 Exposure of dura and middle meningeal vessels.

fascia is separated from the temporal muscle by sharp dissection and turned backwards and forwards as two small flaps The small horizontal incision above the zygomatic arch, shown in Fig 135 is important if adequate access is to be obtained to the base of the skull The temporal muscle is split for the whole

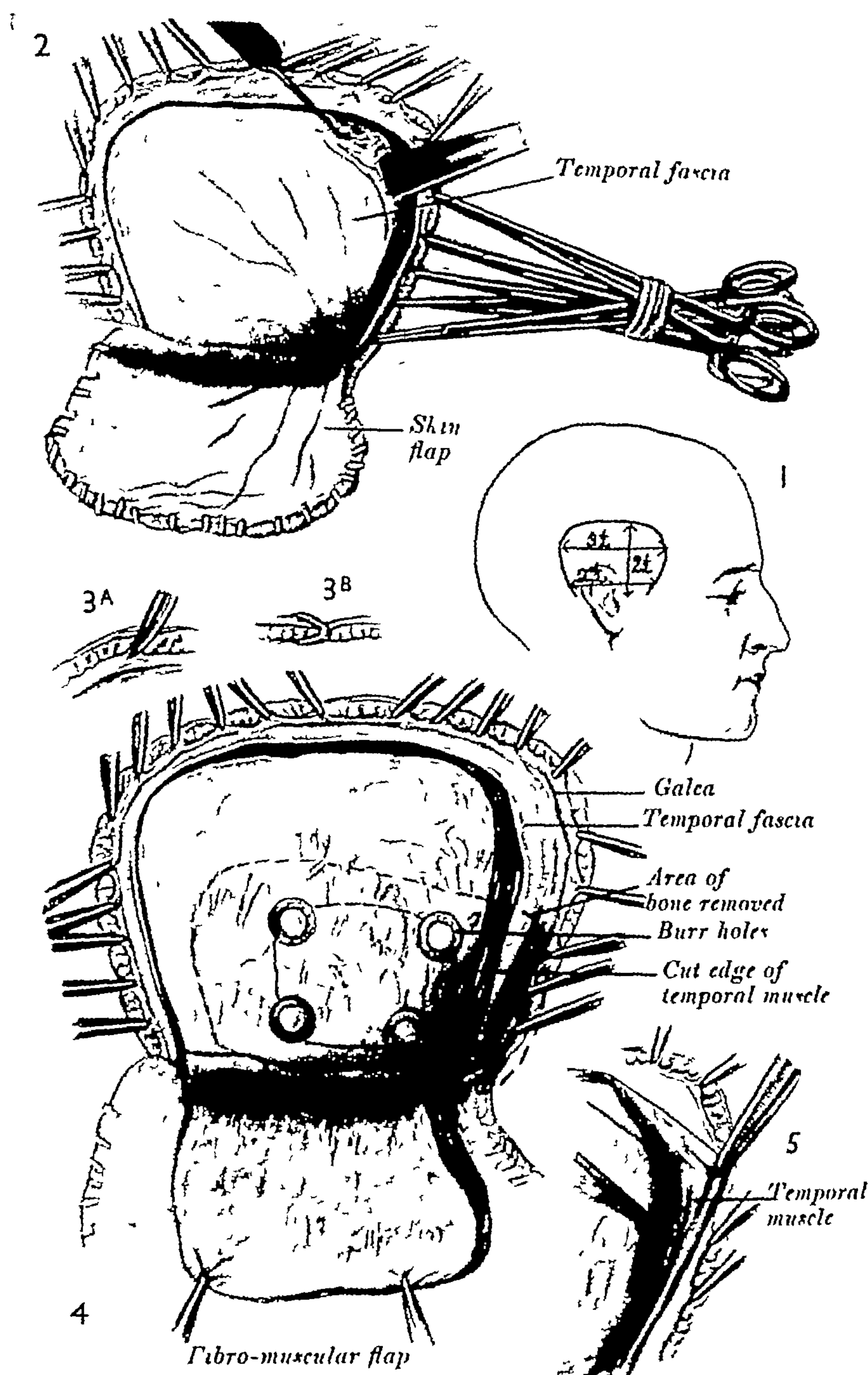


Fig 137

Subtemporal decompression by a muscle sli

1, Line of skin incision and zone of local anæsthesia
2, Subperiosteal separation of the temporal muscle from the bone after the skin flap has been reflected
3A, Method of securing hæmostasis in the skin by hæmostatic forceps clamped to the galea
3B, Method of securing hæmostasis in the skin by Michel clip
4, Proposed area of bone removal after reflection of the skin and temporal muscle
5, Undermining of the anterior and attached part of the temporal muscle to permit of removal of the bone overlying the tip of the temporal lobe of the brain

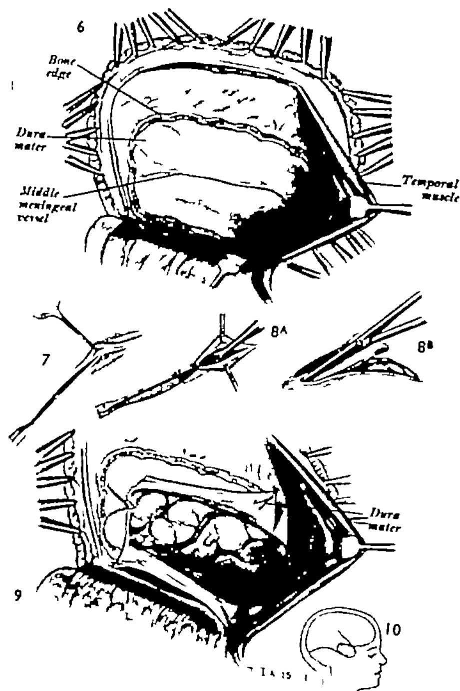


FIG 138

Subtemporal decompression by a muscle slide.

6, Completion of the bone removal. 7 Preliminary incision of the dura mater

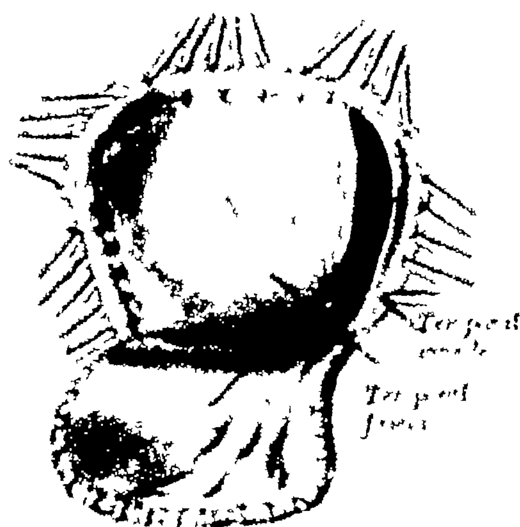
8A Method of enlarging the dural incision by means of a scalpel with a curved dreector to protect the cortex.

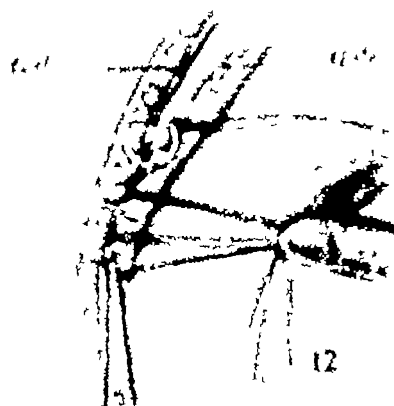
8B, Method of enlarging the dural incision by means of scissors with a strip of wet lintme to protect the cortex

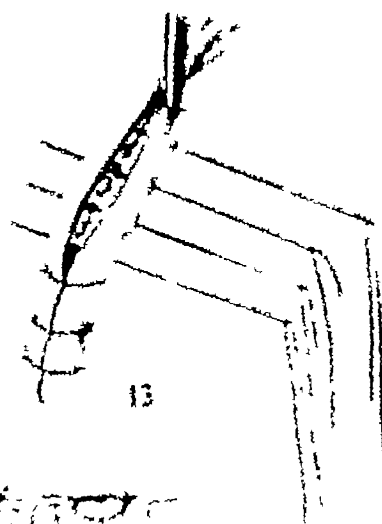
9 Brain decompressed with dura mater sutured to the pericranium to prevent formation of post-operative clots due to oozing from meningeal vessels.

10 Inset shows area of bone which must be removed if the decompression is to act efficiently

length of the exposure in the line of its fibres and then is freely separated from the bone with a curved dissector. One or two









[illegible]

11. [illegible]
12. [illegible]
13. [illegible]
14. [illegible]

self-retaining retractors are now inserted to hold the muscle apart and an area of bone about 1½ in. in diameter should be exposed. A burr hole is made, and after the dura mater has been pushed away with a curved dissector the bone is removed by

nibbling forceps in the whole of its exposure down to the base of the skull (Fig 136) The technique of opening the bone is described below

The Operation of Subtemporal Exploration by Muscle Slide (Figs 137 140)—The incision is shaped like a horseshoe It starts at the middle point of the upper margin of the zygomatic arch and runs upwards and forwards for 2½ in then it curves backwards for 3½ in and finally runs downwards and forwards to end behind the ear on the base line Novocaine is injected along the line of the incision and along the base of the flap from the external angular process of the frontal bone to a point 1 in beyond the posterior end of the margin of the mastoid bone The whole of the lower part of the temporal muscle is infiltrated down to the bone

Digital compression on both sides of the wound is made by the assistant to control bleeding while the incision is made in sections beginning with the skin and deep fascia. Artery forceps or Michel clips are applied as in the previous operation The temporal fascia is now incised ¼ in within and parallel to the skin incision, and the fibres of the temporal muscle in the anterior vertical limb are split down to the bone. The fibro-muscular flap is separated from the bone with a sharp rongeur and turned downwards with the skin flap Sufficient bone is then removed to give adequate access to a bleeding meningeal vessel or to allow for a decompression

The Method of opening the Bone (Fig 140)—The disc at the top of the Hudson's brace is placed in the palm of the left hand and the handle is taken between fingers of the right hand With the perforator screwed in position a funnel shaped hole is made into the bone until the pale blue of the dura mater or the dark blue of an extradural clot is just seen to continue further is dangerous as the dura mater may be pierced and the cortex lacerated The perforator is replaced by burrs of increasing size until a cylindrical hole has been made When using the burrs a certain amount of thrust from the left arm is necessary but the shoulder muscles must be so locked that should the bone give way suddenly the brace is under control and is not allowed to plunge into the brain. It is important to stop turning the brace when the bite on the burr increases suddenly as this means that the inner table has been pierced A thin flake of bone is usually left at the bottom of a burr hole but this is easily pulled or scraped away The next step is to push the dura mater gently from the bone with a curved dissector so that it is not torn when the nibbling forceps are inserted It is much safer to bite the bone away cleanly with forceps than to break it by twisting and leverage, because of the

danger of fracturing the bone beyond the limits of the proposed removal and because the forceps may break or slip and contuse the brain. In the limited exposure of the muscle-split operation one

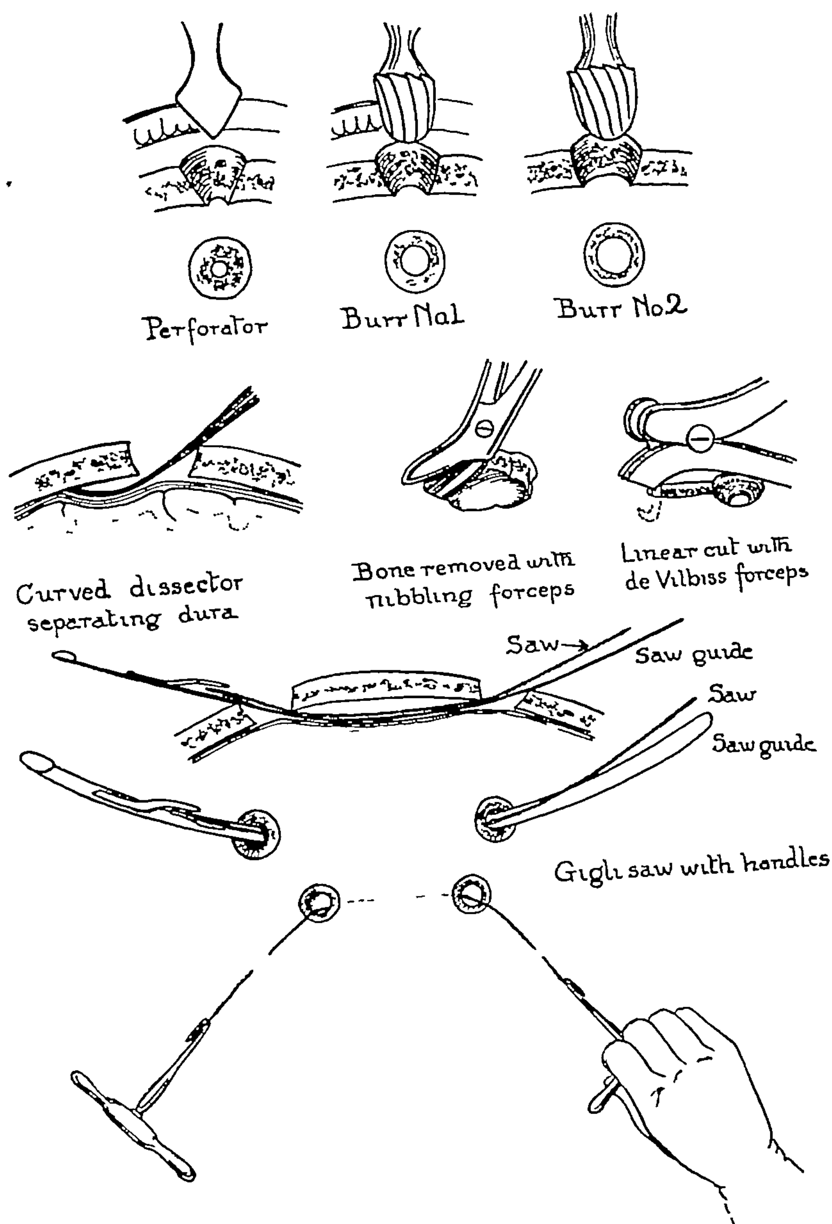


Fig 140

How to open the bone

burr hole only is necessary, followed by nibbling In the wider exposure of the muscle-slide operation four holes are made and linked up by piecemeal nibbling. An alternative method is to join up the holes by linear cuts either with a de Vilbiss forceps or with a Gigli saw. The bone is removed in the lower half

of the exposure, including the part under the attached portion of the temporal muscle It is essential that bone should be removed to the base of the skull if a decompression is contemplated

How to deal with Extradural Hæmorrhage.—Extradural hæmorrhages are usually found in the form of large clots, and very little further bleeding takes place as long as they are left undisturbed When the bone has been removed it is wise to wait for a few minutes to allow the cerebral circulation to readjust itself to the new conditions It is extremely dangerous to insert a finger into the wound in an endeavour to hook out the clot, as troublesome bleeding may be induced before one is in a position to control it. Moreover, the extra pressure of the finger superimposed on the already existing compression of the clot may prove fatal The clot should be removed slowly a little at a time from above downwards with a suitable instrument, such as a teaspoon or a curved dissector until the ruptured meningeal vessels come into view The bleeding vessels are most easily sealed with the coagulating diathermy current, but if this is not available they may be underrun with a fine suture and ligatured or compressed with a silver clip The vessels rupture usually at a point above the base of the skull and are easily accessible. Rupture on the base of the skull is much less common and the necessity of plugging the foramen spinosum with a wedge of matchstick or with bone wax to stop bleeding is fortunately rare. To do this successfully an efficient headlight is essential and the temporal lobe must be raised with flat brain retractors

Profuse bleeding which seems to come from many angles is usually not due to rupture of the meningeal vessels, and its control often presents most difficult technical problems It may come from diploic veins in a fractured bone or from a ruptured venous sinus Horsley's wax should be pressed firmly into the crack of any fracture that can be felt or seen

When in spite of careful waxing bleeding continues and streams from above it is probably coming from the superior longitudinal sinus

In such cases even though direct viewing of the sinus through the subtemporal exploration is impossible it is unwise to make a further opening near the midline to try and expose the tear since its exact location is not known It is usually safer to cut small grafts from the temporal muscle and to pack these towards the bleeding point between the dura mater and the bone with a pair of long dissecting forceps When this has been done small stitches should then be passed through the dura mater at the upper edge of the exposure and taken over the bone and through the

pericranium, so that when the sutures are tied the drag on the dural envelope will keep the grafts in position When the bleeding comes from the base, muscle grafts should be packed towards the petrosal, sphenoidal and cavernous sinuses. Occasionally it may be necessary to use strips of gauze instead of muscle grafts. When this is done they should be removed forty-eight hours later under direct vision with the wound widely open. If they are withdrawn blindly through a small opening, serious bleeding may start again.

Opening of the Dura Mater for Decompression of the Brain.—When the brain is under tension, opening of the dura mater can be a very difficult manœuvre, and the greatest care must always be taken not to bruise the brain or wound the cortical vessels. The opening is made as high as the Sylvian fissure but no higher, otherwise the lower end of the motor cortex will be endangered. On the other hand, it must go down to the base of the skull if the decompression is to act efficiently. A cross-incision will be found most useful in the muscle-split operation, but in the wider exposure produced by a muscle slide a rectangular or semicircular flap may be turned upwards. With a sharp scalpel an incision ½ in. long is gradually deepened until a curved dissector can be inserted between the arachnoid and dura. The incision is then enlarged by cutting down on to the blade of the dissector An alternative method of continuing the incision is to cut the dura with scissors after direct vision has been obtained by depressing the brain for a few millimetres with a strip of wet lintine pushed into position with a pair of narrow-bladed dissecting forceps (see Fig. 138)

Methods of controlling Bleeding (Figs. 141 and 142).—As the circulation of the brain is already embarrassed, loss of blood during an operation is dangerous. Care, therefore, must be taken to have any possible bleeding under control before any manipulation in an operation is begun, and in particular, digital compression of the skin must never be omitted as the first incision is made.

A coagulating diathermy current is used for sealing veins or small arteries in the muscle, dura and brain. The neatest and most convenient method is to pick up the vessel concerned between the blades of a finely pointed dissector, and as the field is dried with the sucker, to touch the dissector with the diathermy electrode. A warning, however, is necessary, for although the diathermy current is so useful in controlling bleeding in the deeper tissues it must never be used on or superficial to the galea, otherwise the skin edges will necrose and the wound will not heal by first intention Care must also be taken that the artery

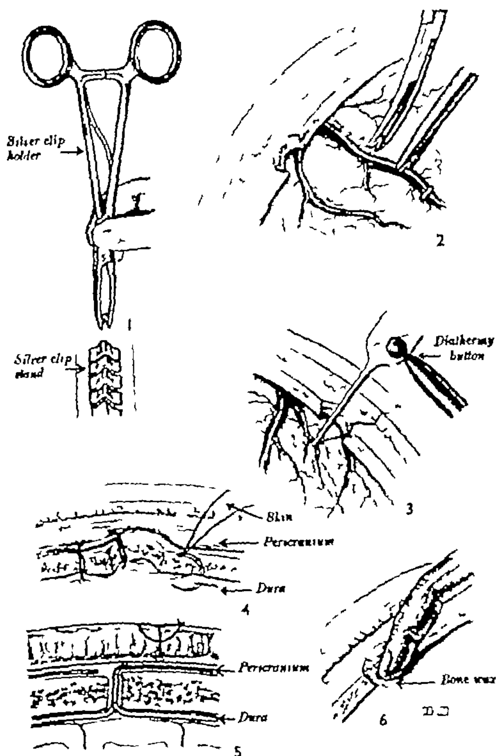


FIG. 141

Methods of controlling bleeding.

1 and 2, Compression by silver clips. 3, Diathermy coagulation.
4 and 5 "Hitching" of dura to pericranium to prevent extradural clots.
6, Plugging of diploic vessels with Horsley's wax.

forceps are not touched inadvertently, otherwise the same complication will occur.

When a large artery is bleeding or has to be divided, it is safer to compress it with a small silver clip applied on a special holder. Ligature of the delicate cerebral vessels is always a lengthy process and often difficult.

Hæmorrhage from bone is easily controlled by pressing Horsley's wax firmly into the bleeding channel.

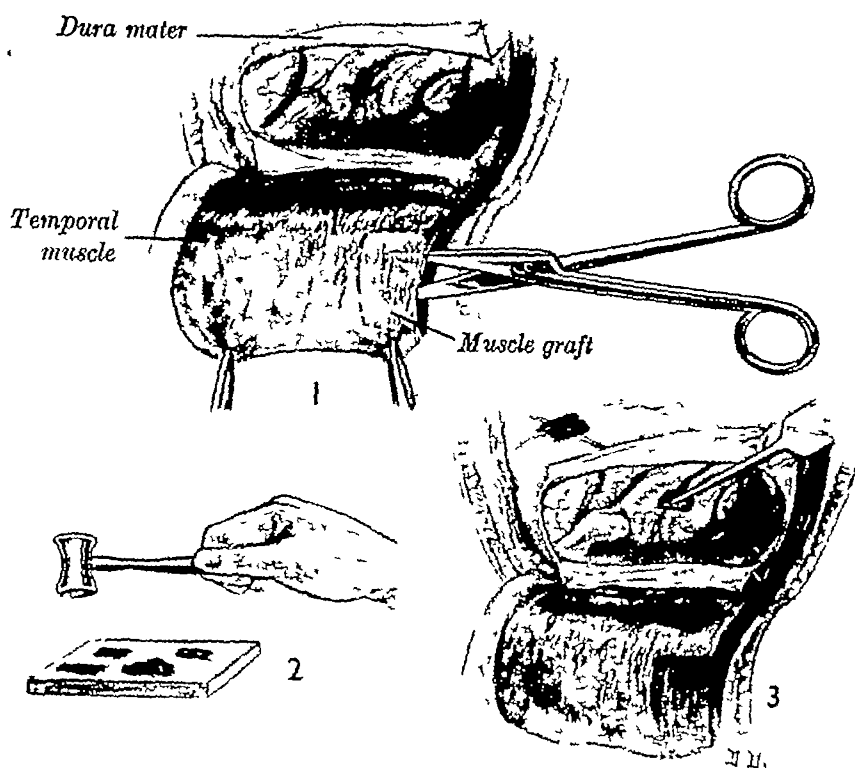


FIG 142

Preparation and application of muscle grafts

1, Cutting graft from temporal muscle The whole thickness of muscle is not used
2, Hammering of muscle grafts into thin sheets
3, Grafts in position on the cortex and dura

Muscle grafts are of particular value in the repair of tears in the venous dural sinuses. They are also useful in controlling persistent bleeding from the surface of the brain when other methods have failed. A flat piece of muscle cut from the temporalis and hammered into a fine sheet will stick like a postage stamp when placed in position.

Before any wound is closed it must be left completely dry as far as bleeding is concerned, otherwise a post-operative clot will form It may be taken as an axiom that a bleeding cerebral vessel,

however small, will continue to bleed and compress the brain before the tension in the clot rises sufficiently to occlude the bleeding point. Many post operative hæmorrhages can be prevented by tacking the dura mater at the periphery of the wound to the pericranium with occasional interrupted sutures. Thin layers of clot on the dura itself are best left in position since they are unlikely to give rise to compression when the wound is closed. Also, they act as seals to numerous small vessels which otherwise would bleed.

Wound Closure—*Muscle Split*.—The edges of the muscle are apposed in their whole thickness by interrupted sutures of fine silk or thread introduced on small curved needles.

The method of suturing the temporal fascia depends on whether or not the dura mater has been left opened. In decompressions the muscle only is drawn together so that its fibres can, if necessary, stretch apart and accommodate a bulge of the brain. When the dura mater has not been opened the fascia may be sutured, but as it often contracts during the operation it is usually impossible to repair more than its lower half.

Muscle Slide.—As the temporal fascia will slide to some extent across the muscle face, any natural retraction of the membrane during the stages of the operation can be easily overcome by the following manœuvre and an excellent covering for the bony opening obtained. The fascia is sutured above and behind but left open in front; here, only the muscle fibres are drawn together (see Fig 139).

Buried sutures introduced at intervals of ¼ in. are used to appose the galea. These sutures must never be omitted or replaced by deep through and through sutures, because the integrity of the wound depends on this layer. Care must be taken to cut the buried sutures accurately on the knots with fine scissors, otherwise long ends of silk will get between the skin edges and prevent healing. Correctly inserted galeal and skin sutures will control superficial vessels, and it is unnecessary to ligate each individually (see Fig 139).

When a wound is not under great tension the stitches may be removed in three days.

A sterile gauze dressing is placed over the wound and fixed in position by winding a sterile 6-in. gauze roll around and over the head. In a restless patient, when there is a danger of his removing the dressing or of putting his fingers on to the wound, a stout linen bandage is also used. This is fixed under the chin, but never wound round the neck because of the danger of compressing the internal jugular veins and thus causing cerebral venous congestion with renewed bleeding (Fig 143).

Convalescence.—Convalescence starts with the return of consciousness.

When should a Patient be allowed to get up and out of Bed.—This is an important question. The answer is as soon as he is desirous of so doing and is reasonably able, provided that he is properly orientated regarding time and place Prolonged rest in bed commonly leads to psychoneurosis When a patient has not been concussed a linear fracture of the skull is of no particular significance and a patient needs only to remain in bed for one or two days. On the other hand, it is wrong to hurry a patient out of bed until he shows some desire to do so. In particular, he should not be allowed to roam about the ward when obviously confused The danger of allowing a patient out of bed too early is that should symptoms of the post-concussional syndrome develop later they are apt to be attributed to inadequate rest and treatment in the early stages. Patients are often heard to say that if they had taken enough care in the early stages they would not have suffered later.

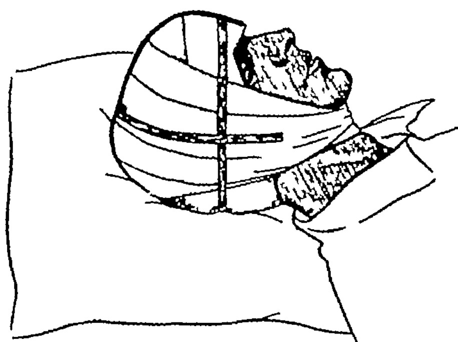


FIG 143

Method of securing a dressing on a head wound (*Norman Dott*)

After a severe head injury three weeks may be allowed for convalescence.

The first week should be passed extremely quietly, reading or listening to the wireless being forbidden When relatives are worried about a patient's mental state the importance of quiet should be fully explained to them, and they should be assured that improvement will almost certainly come in time Anxiety, and in particular domestic worries, must be avoided Tactful reassurance of the patient materially assists recovery During the second week the patient is encouraged to move freely, his limbs are exercised and he should be occasionally lifted into an armchair when his bed is being made He may be allowed to read or listen to the wireless and talk with his fellow-patients if he wishes. A light but nourishing diet must be given and his bowels kept freely open with mild aperients. Headaches, dizziness, insomnia and depression are common symptoms; if they are belittled or overlooked, the confidence of the patient may easily be lost, since he will form the impression that the surgeon does not understand his case. On the other hand, if symptoms are given too much attention, a troublesome functional overlay may result. Careful judgment, therefore, is necessary on this score on the part

of the doctor in charge. It is very demoralising and frightening to a patient at this stage to give him the impression that he has been very seriously injured and is lucky to be alive. Also, it is a bad psychological error to discuss his case at the bedside and to give pronouncements on the X rays. A patient attributes something very sinister and crippling to a fracture of the skull which in itself may be of very little importance. Medical argument is apt to make a patient untrusting and apprehensive that his ailment is not properly understood.

Head pains may at first be exceedingly severe but will usually subside under correct treatment. Whether the head is kept low or raised on pillows depends entirely on the position that gives the most comfort. At this stage the cerebrospinal fluid pressure must be measured to guide treatment. As it is not often high, withdrawal of cerebrospinal fluid or intravenous dehydration will rarely give relief. When the pressure is subnormal the head should be kept low and copious fluids given by mouth, so that a little more is taken than is actually needed to quench thirst. By these means an increase in the secretion of cerebrospinal fluid can be encouraged and the intracranial pressure brought back to normal. Aspirin and Veganin may be used freely; small doses of bromides are soothing, and an adequate amount of sleep must be ensured, if need be by drugs such as Nembutal, Dial or Medinal.

In all cases of compound injuries of the brain, and in closed injuries when epilepsy has occurred during the acute phases, sodium phenobarbitone (½ gr.) should be given night and morning to reduce the tendency to convulsive seizures.

The third week is an important period. Each day the patient is made to get out of bed for increasing periods; his muscles are toned up by gradual exercises until he can walk, and he is taught to bend and to stoop in an effort to restore his vasomotor control.

Period of Rehabilitation.—At this stage in his recovery the patient is allowed to leave hospital, but strict instructions must be given if he is to improve during the following weeks. He must not be allowed to develop the habit of staying in bed in the mornings: he must have breakfast at the normal hour and only then should he be allowed to rest if he finds it absolutely essential. Each day for a fortnight he should go or be taken into the fresh air and should be firmly discouraged from huddling before the fire for long periods. Then at least two weeks should be spent at the seaside and the general health improved by a tonic of neurophosphates. Regular habits should be aimed at.

Return to work will now have to be considered. Unfortunately this problem is often complicated by the question of compensation

Immediate return to full or arduous duties is always undesirable, because the patient is apt to break down under the strain, and if he should do so his loss of confidence may initiate intractable neurasthenia. For example, it is most unwise to allow a middle-aged man who has been severely concussed to resume heavy manual labour or work amongst noisy machinery. On the other hand, a patient should be encouraged to adapt himself to his new physical state, and much economic waste could be avoided if really suitable employment could be found. The problems of rehabilitation and compensation will be discussed in Chapter X.

CHAPTER V

FRACTURES OF THE SKULL

THE skull is developed from the plate of mesenchyme which surrounds the cephalic end of the notochord. The cranium is at first entirely membranous and forms a capsule for the cerebral vesicles. Only the base of the primitive membranous cranium undergoes chondrification. The parts of the skull which ossify in membrane are as follows :—

1. The tables, or upper portions, of the occipital bone.
2. The two parietal bones.
3. The frontal bone.
4. The pars tympani and the squamo-zygomatic portion of the temporal bone.

At birth the bones of the skull are separated from one another by fibrous tissue which is continuous with the periosteum externally and with the dura mater internally. The skull rapidly increases in size during the first six years and reaches its final dimensions about puberty. The paranasal air sinuses begin to develop just before puberty, although, surgically, it is of importance to know that they are present and may even be large at an earlier age. In old people they become considerably enlarged.

There are individual as well as racial differences in the shape of the skull. The dolichocephalic head is long and narrow (Fig. 144, A, D), the brachycephalic head is short and broad. The normal cubic capacity of the cranium lies between 1,450 and 1,500 c.c. At the two extremes lie the enlarged or hydrocephalic and the small or microcephalic skull. Thickness of the individual bones of the skull differs considerably both in health and in disease (Fig. 145). It will be appreciated, therefore, that apart from the variable factors of the injuring force—magnitude, direction, site and area of application—the skull itself has factors which differ with each individual, *i.e.* size, shape and toughness and elasticity of the bone. Moreover, the head in many accidents is subjected to more than one violence, and the point of reaction and duration of the force of reaction may vary. Thus it is not

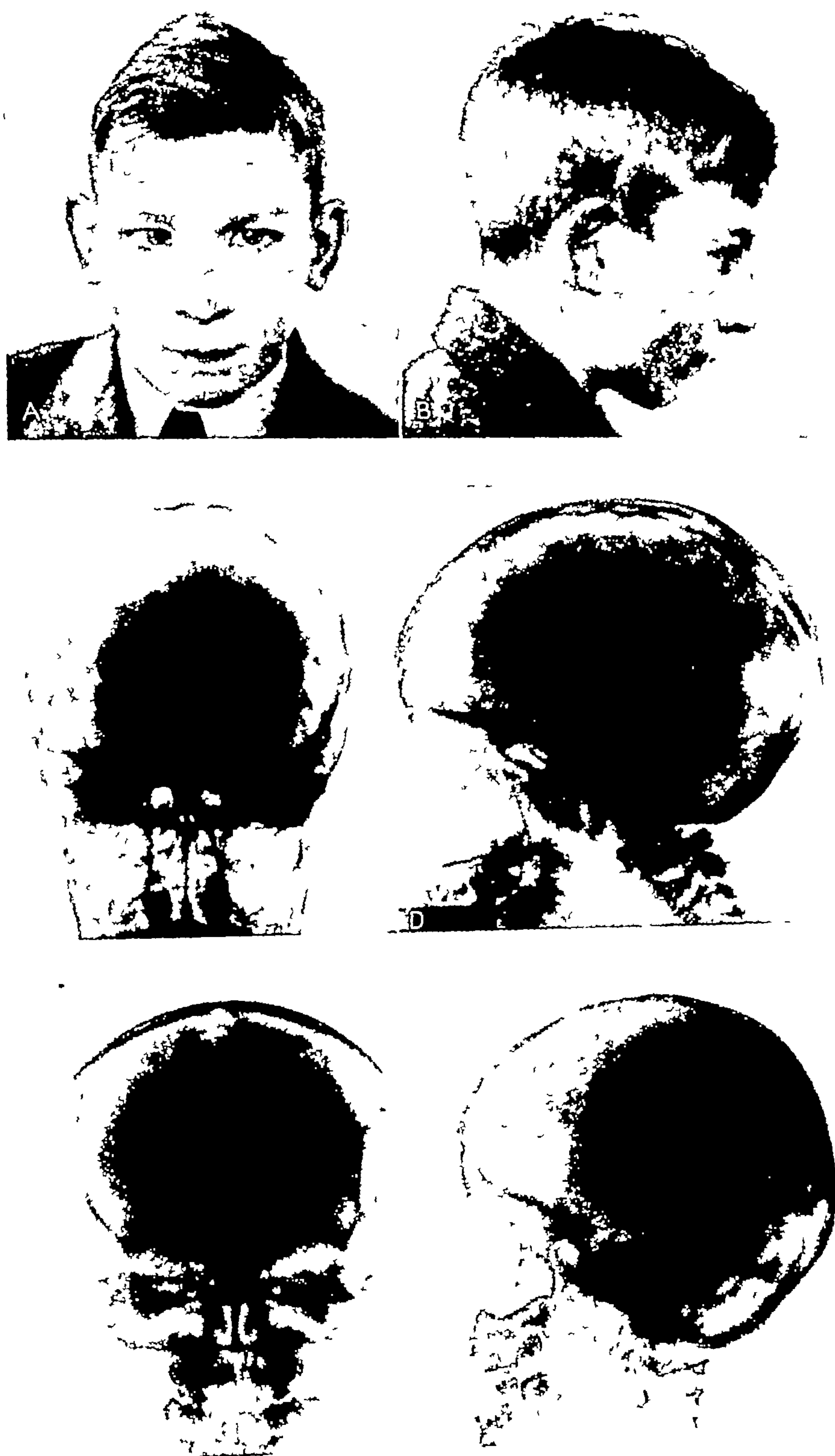


FIG 144
A dolichocephalic skull (narrow and long) will behave differently from a brachycephalic skull (short and wide) when subjected to trauma

surprising that fracture patterns are so variable. The following types of closed fractures are encountered—Linear, Indented and Depressed

LINEAR FRACTURES

Linear fracture lines may be short or long and they may run in all directions over the surface of the skull. They may stop and then start again a short distance away, the continuing line



Fig. 143

X ray of the skull of a child aged two and a half years. It will be readily appreciated how bone as thin as this can be indented or fractured.

running in the same direction as the initial line, or in a different one. Commonly a linear fracture has a stellate pattern. The fracture lines may be part of a depressed fracture and run radially from the periphery of the depression. They may be confined to the base or to the vault or as so commonly happens they involve both the vault and the base. As a rule there is no displacement, however when this occurs it usually takes the form of widening without depression and is more often seen in children than in adults. Linear fractures may run into the foramen magnum or

across the optic foramina or into any of the foramina through which the cranial nerves pass as they emerge from the cranial cavity on to the face or neck. Or, again, they may open into the paranasal air sinuses or the middle ear, in which case they cause internal compounding; these latter fractures will be discussed later. Linear fractures are so variable in extent, number and pattern that any kind of detailed classification is profitless (Figs 146-150).



FIG 146
Springing of the fronto parietal suture is equivalent to fracture

Treatment.—For undisplaced linear fractures of the closed type no surgical treatment is necessary. Within a few weeks a fracture line is filled with fibrous tissue, and for most purposes the protective efficiency of the skull is not materially impaired. Despite statements to the contrary, most linear fractures heal by bony union. This takes about twelve weeks in a child and from one to three years in an adult. When fractured surfaces are separated by more than a few millimetres, fibrous union is the rule, but even in these cases isolated strands of bone often form and radiological evidence of fracture disappears. "Springing" of



FIG. 147

A linear fracture of the circular type in the transverse plane associated with a depressed fracture



FIG. 148

A horseshoe shaped fracture of the frontal and parietal bones



Fig 149

A linear fracture running into the frontal air sinus causing internal compounding with the danger of meningitis



Fig 150

A basal shoot will occasionally reveal a linear fracture running across the ethmoid bone and presumably opening into the air sinus This is an internally compound facture

a suture is equivalent to a linear fracture and is often seen at the occipitoparietal junction Spicules of bone projecting from the inner table for more than ¼ in should be removed This is done by cutting out the affected segment of bone with a trephine of suitable size The spicule is then removed by a chisel or nibbling forceps and the skull repaired by hammering the bony disc back into position *It is unnecessary to keep a patient with a simple fracture of the skull in bed for more than a few days Indeed it is a mistake to call undue attention to its presence, owing to the danger of encouraging some kind of troublesome functional overlay* Occasional observation however, is necessary in order to make sure that an extradural clot does not form and compress the brain

The main difficulty in this type of case is to decide when a patient may take up his ordinary mode of life Recently I was asked to see a famous soccer internationalist who had received a linear fracture of his frontal bone without displacement. He was a vigorous forward highly skilled in using his head to any kind of ball A tribunal decided it was unwise to let him play again My opinion is that when radiology shows a fracture only as a fine line, a man may be allowed to play either association or rugby football at the end of a year whether the fracture heals by fibrous or bony union When a fracture is extensive and its opening is more than 3 mm in width, it is unwise to give medical permission for such exercises, even though the danger of injury consequent on the weakness of the skull is exceedingly small It is of course, quite justifiable to tell the patient that his skull is not seriously weakened and will withstand considerable violences without harm A linear fracture does not prevent a man from resuming his occupation even if he is a miner or quarryman, and it certainly does not detract from the efficiency of a member of any of the Services

INDENTED FRACTURES

Indentations may be pointed or rounded They occur chiefly in children when the bones are thin and plastic and often are not associated with a demonstrable fracture line At birth, particularly following forceps delivery, rounded and large indentations are often seen in the skull Most of these disappear within the first few weeks as the child s brain develops, and need cause little anxiety The indications for surgical treatment are as follows —

1 *At Birth*—Large indentations associated with signs of cerebral compression
2 *At Three Months*—(*a*) Disfiguring indentations outside the hairline (*b*) Any indentation over the motor cortex.



Fig 149

A linear fracture running into the frontal air sinus causing internal compounding with the danger of meningitis



Fig 150

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(c) Indentations associated with symptoms such as epilepsy or obvious mental deficiency.

3. *At Three Years.*—All indentations irrespective of their position when more than 1 in. in diameter.

Methods.—A small trephine hole is made at the periphery of the indentation. A suitably curved dissector is then passed through the operative opening and used as a lever to raise the depressed bone. At the end of the operation the trephine disc is replaced so that a defect is not left in the skull. When this method fails owing to loss of plasticity of the bones, Dott has suggested that a hole should be made through the bone at the apex of the indentation



(a)



(b)

FIG 151

(a) Before operation (b) After operation.

and radial cuts made to the periphery. This allows each sector of bone so produced to be bent outwards at its base and moulded to the normal contour of the skull.

DEPRESSED FRACTURES

Depressed fractures of the closed type frequently involve large areas of bone and are caused by the head being struck over a broad surface. As in open fractures, they are often comminuted, although the affected bone is rarely broken into small fragments Occasionally a complete bone is loosened at its suture lines and displaced not only inwards but sideways, so that one of its edges slides between the dura and intact skull.

Closed depressed fractures, even when extensive, are frequently unassociated with signs of local brain damage, and when a patient is conscious it is often difficult to decide whether their elevation by operation is necessary or not. When they are not causing obvious symptoms or signs and there is danger in raising them,

as, for example, when they overlie the sagittal sinus, they are better left undisturbed. The decision to operate, of course, will be influenced by the age of the patient and his type of work. A weak skull in a schoolboy is obviously a serious disability not only because it precludes him from playing games but it may seriously affect his chances of obtaining employment later in life. In such circumstances it is better to operate. On the other hand, when a man is living a sedentary life and has family responsibilities, it is wiser not to operate.



Fig. 15.

A lateral view of a fractured skull showing a depressed fragment of bone end on. Such an X ray picture means that the depression of the bone is considerable and that the dura is almost certainly lacerated.

The specific indications for operative treatment are as follows:—

1. When a patient is unconscious and thought to be suffering from cerebral compression.
2. When there are signs of underlying brain damage, such as hemiplegia or aphasia, or when a patient has persistent symptoms of headache and giddiness.
3. When a fragment of bone is thought to have pierced the dura. This judgment depends on the shape of the bony fragment, on its angle of tilt and on the amount of depression. Fragments depressed for more than ½ in.,

those lying end on, or those obviously spiculated should be elevated (Fig. 152).

4. Cosmetic considerations are important not only in women but also in men. Depression and loss of confidence may often be traced to an inferiority complex caused by brooding over a physical disability which is noticeable to other people. This functional complication should never be overlooked, as it can often be corrected by repairs of the skull.

Treatment.—Correct positioning and shaping of the skin incision for the exposure of an extensive cranial depression is very important; often it is a difficult manœuvre. Certain basic principles have to be observed

1. The incision must be so designed that the blood supply of the skin edges are not jeopardised. Patients with healed but complex scalp wounds are now coming into hospital from the battlefields, and cranial defects have to be repaired, foreign bodies removed and meningo-cerebral scars excised. The difficult problem is the extensive stellate scar. In these cases if the usual semicircular flap is employed, the transverse scar of the original wound cuts off the blood supply to the dome of the flap and may cause imperfect healing or even extensive sloughing. This difficulty may be overcome by incising along one line of the scar and by extending the incision on to normal skin at each end. If the incision be made long enough even the largest depressed fracture or cranial defect can be adequately exposed (Fig. 153). Every effort must be made to avoid opening three or more limbs of a stellate scar, because healing at the apex is apt to be delayed if this is done

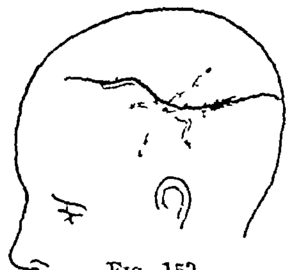


FIG 153

When a stellate scar is present a cranial defect can be adequately exposed by opening one limb of the scar and extending the incision into normal tissues

2. The cranial exposure must be adequate An inadequate exposure always leads to unnecessary technical difficulties and often to imperfect workmanship. This is a fault liable to be made by the inexperienced.
3. The exposure of the bone must be such that a correct bone flap can be raised on those occasions when it is necessary to excise a meningo-cerebral scar.

The raising of a large fragment of bone is not a simple operation and cannot be effected by leverage through a trephine hole. It

necessitates exposure of the whole of the depressed fragment by means of a suitably designed skin flap The type of operation necessary depends on the findings after the skin flap has been raised

When the depressed fragments are loose and move easily all that is required is to lift them and to arrange them in position They may be anchored by sutures passed through the pericranium covering them This however, is generally unnecessary, as they are usually held firmly enough in position between the dura and scalp as the skin flap is closed Completely loose fragments without blood supply will readily consolidate and no misgivings need be entertained on this score Also tight fitting as in grafting in other bones, is not essential or even important.

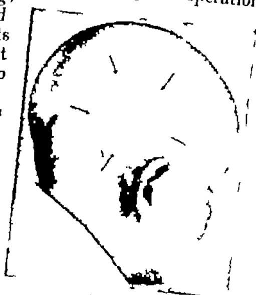


Fig 134
Treatment of a depressed fracture of the skull
Clinical photograph showing the indentation.

A block resection of depressed fragments is necessary when they are tightly interlocked and cannot be disentangled As

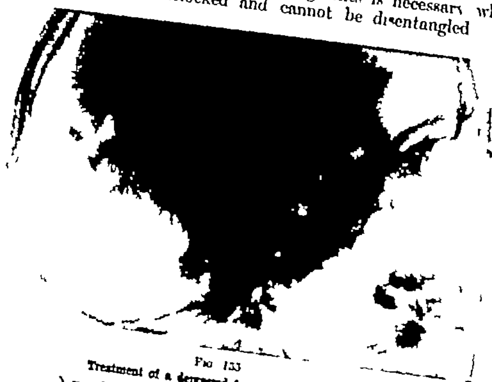


Fig 133
Treatment of a depressed fracture of the skull
X-ray of the depression giving lateral view of indentation.

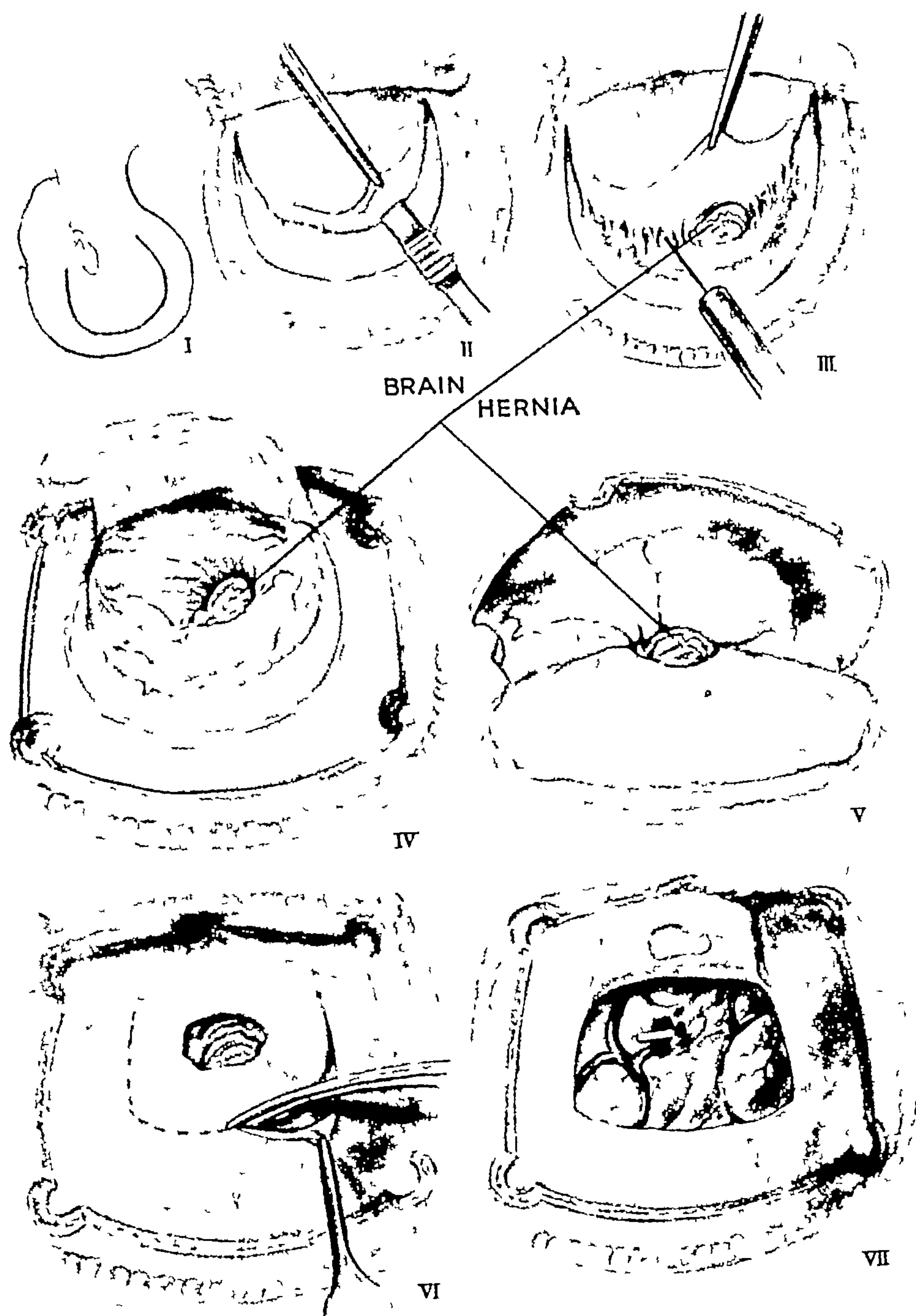


Fig 156

Treatment of a depressed fracture of the skull

I Line of incision
II }
III } Reflection of temporal muscle and fascia
IV }

V Raising of bone flap
VI Opening of dura
VII Exposure of cerebral hernia and laceration

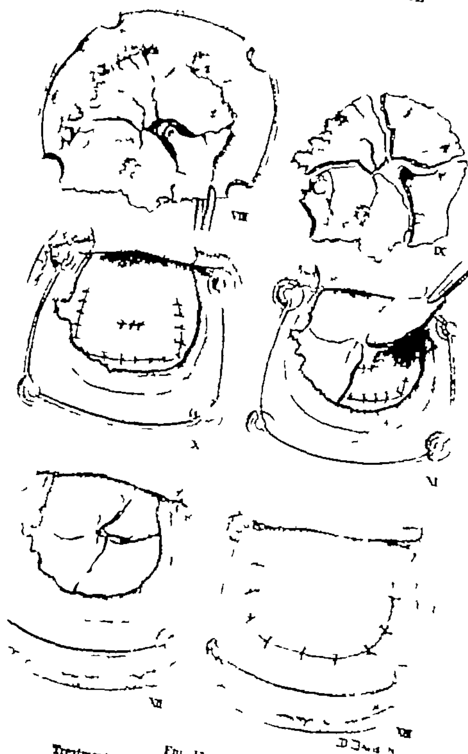


FIG 13

Treatment of a depressed fracture of the skull

VIII Depressed fracture seen from under surface after block resection.
IX Fracture broken into component part
X Repair of dura mater
XI Replacement of bone fragment
XII Final build up after depressed bony fragments have been raised.
XIII Repair of temporal fascia and muscle

described in the next chapter, a block of bone is removed in these cases and the displaced fragments hammered into position The remoulded block of bone is then replaced and anchored by means of silk or fine wire sutures passed through drill holes made in suitable and corresponding positions in the block and surrounding skull Often suture of the pericranium of the graft to that of the surrounding skull will suffice to keep the graft in position until it consolidates. In old-standing depressed fractures, when the bony fragments have united by fibrous or bony union, the bone after block resection may be reversed so that its inner surface faces outwards if the contour of the skull will allow When this is not possible, the fragment has to be broken up by the best means available at the time. No set operation can be described for these cases, as the conditions are so variable Each case, therefore, has to be judged on its merits, the actual corrective manœuvre depending on the surgeon's skill and ingenuity (Figs. 154-159)



FIG 158

Treatment of a depressed fracture of the skull

XIV After repair

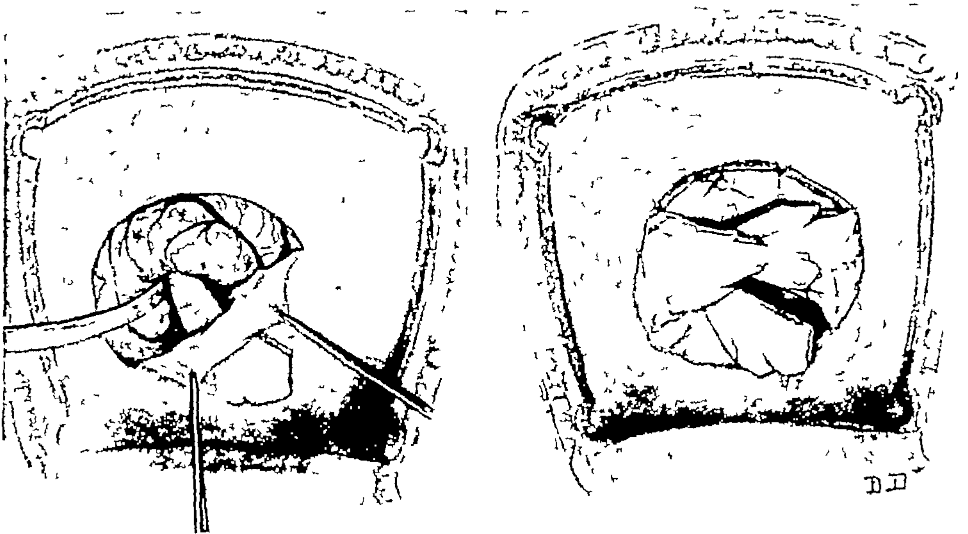


FIG 159

When a bony fragment has been driven deeply into the cerebral tissue it is much safer to remove it under direct vision rather than to rock it out blindly

DEFECTS IN THE SKULL

The most common cause of defects in the skull is the operative removal of bone fragments in the débridement of compound depressed fractures. Less frequently, defects are the result of explorations and decompressions made for the evacuation of extradural clots or for the relief of pressure in cerebral œdema. Loss of bone in road and industrial accidents caused by an injuring force is extremely rare and is no more common in air raid casualties. It does, of course, occur more often in gun shot wounds but many of these are fatal. In children, skull defects occasionally result from destruction of an epiphysis by injury of the closed type. This leads to disappearance of bone in a wide area as shown in Chapter II, Fig. 48 (p. 56), and is known as traumatic malacia.

The disability caused by a calvarial defect is not necessarily serious, and depends on its size, position and the part it plays in the production of post traumatic epilepsy. A defect naturally is much more serious when the underlying dura is open and the brain is adherent to the scalp than when the dura is intact, as drag on the cerebral cortex is so prone to cause headaches, dizziness or epilepsy. It is often difficult to decide, particularly in the absence of symptoms, whether or not a repair of the skull is necessary, and it will be helpful to keep the objects of treatment clearly in mind. These are fivefold —

(i) *For the Cure or Prevention of Traumatic Epilepsy.*—As the cause and treatment of traumatic epilepsy will be discussed in a later chapter, all that need be said here is that according to circumstances, adequate repair of the skull may be an important factor in its cure.

(ii) *For Protection of the Brain.*—In young people, repair of a defect in the skull is necessary if their normal activities such as the playing of games are not to be restricted. This is an important consideration and parents will usually give consent to an operation when the arguments for and against repair are presented to them. A weak skull is obviously a serious disability in miners or industrial workers whose heads are liable to be knocked on account of the nature of their work. Correctly fitting protective helmets or splints will give adequate protection under these conditions but often a workman refuses to wear them or finds them uncomfortable or too hot to be tolerated for long periods. When objections to external

protective methods are made, operative repair of a defect is fully justified and usually gives excellent results. In members of the Services a calvarial defect usually leads to discharge or to regrading to a lower physical category This is often unnecessary, since a simple bone graft would make the man concerned just as efficient as before.

(iii) *For Cosmetic Purposes.*—Disfigurement cannot be measured by scientific standards It is purely a matter of personal opinion, and the only point of importance is the opinion of the patient himself or, in the case of a child, that of the parents. If the appearance of a defect is worrying to a patient, then it should be repaired, wherever or however small it may be Very few people will overlook the unsightly deformity caused by the loss of a supra-orbital ridge, particularly if the brain slips forward and bulges over the eye.

(iv) *For Relief of Giddiness and other Symptoms consequent on Instability of the Cerebral Circulation.*

(v) *For Psychological Reasons.*

TREATMENT

General Considerations.—Repair of a calvarial defect must never be considered until a wound is free from infection and the skin soundly healed over it. In the presence of infection a graft will not consolidate and will, in fact, aggravate inflammatory processes by acting as a foreign body and increase suppuration until it is discharged from the wound Unfortunately skin may heal over tissues which are potentially infected. On those occasions, therefore, when the original operation was not done by oneself, it was essential to know how thoroughly débridement was carried out in the first instance before deciding to reopen a wound An unhealthy-looking scar, repeatedly scabbing in spite of treatment, is suggestive of infection and must be regarded as such. After a few months, radiography often will give valuable information regarding the presence or absence of infection, since in a clean wound the margins of the bone will be sclerosed, whereas when infection is active the bony edges may be fluffy or sequestra may show.

As a working rule it is wiser to wait for three months, even in favourable cases, before operative measures are considered, and this period should be even longer in doubtful cases When operative repair of a defect is not advisable and protection is necessary, a metal splint moulded to the shape of the head and held in position by suitable straps is the usual method adopted by instrument

makers Such metal splints are often uncomfortable, and I have found that a felt pad is more useful than a metal plate This can be sewn into the cap or hat, thereby avoiding the necessity of tight strapping to hold it in position

General Operative Technique —A skin incision is designed to expose the whole of the calvarial defect concerned, including the tissues beyond its margins for a distance of at least ½ in On the forehead an incision running vertically in the middle line will heal with the minimum of obvious scarring A frontal defect may be exposed by continuing the upper end of a vertical incision outwards and downwards into the temporal fossa within the hair line, and by turning the skin flap thus produced forwards and outwards (Fig 160)



Fig 160

This type of incision for exposure of a defect in the frontal bone leaves a very inconspicuous scar

After the skin has been reflected the pericranium is usually found to be densely adherent to the dura over the edges of the bony defect. When this occurs, an incision through the pericranium at the periphery of the bony opening will be necessary before it is possible to separate the dura from the bone Such separation often causes considerable bleeding and great care must be taken to have the dissector under control when this manœuvre is carried out, otherwise the dura may be torn and the brain lacerated The object of freeing the dura is to ensure that grafts fit snugly into the defect and to enable holes to be drilled into the bone if the graft has to be sutured into position by this means

Methods of repairing a Bony Defect —The older method of insinuating a piece of tin foil or perforated celluloid plate between the dura and bone has been superseded by the use of bone grafts which may be taken from (1) the external table of intact skull (2) the ilium, (3) the tibia or (4) the ribs. Autogenous grafts are the best but in children bone may be taken from the mother or from a donor of the same blood group

Grafts taken from the External Table of the Skull —A piece of sterile lint or gutta percha tissue accurately fitting the bone defect is cut out and placed over intact skull which, according to the

size of the graft required and to the vascular requirements of the scalp, may or may not need to be exposed through a separate incision An incision is then made through the pericranium ½ in. outside the boundaries of the pattern, and the membrane separated inwards with a rongeur for this same distance so that a flange of pericranium is produced. By means of a circular saw the external table is cut in the shape of the pattern and the



Fig 161
Rib grafts will give an excellent repair of a cranial defect

graft removed by means of chiselling through the diploe. The graft is then fitted into the defect and its flange of pericranium stitched to that of the surrounding skull One of the difficulties of this method is that the graft is apt to splinter when being lifted Also, it is inapplicable to children, in whom the diploic cavity is absent or imperfectly formed

Rib Grafts.—The advantages of rib grafts are that they are easily taken and readily moulded to the contour of the skull (Fig 161). When this method has been decided upon, the lower ribs are exposed in the mid-axillary line and lengths of bone removed subperiosteally according to the size of the defect to be repaired After removal the rib is split longitudinally along its broad plane so that two flat grafts are produced By suitable transverse cuts the bone may be weakened and so bent that it fits the contour of the surrounding skull. Grafts should always be arranged so that their smooth surface faces the dura

An alternative method of cutting a graft is to make incisions through the fibroperiosteum at the upper and lower edges of the rib instead of centrally, as in purely subperiosteal resections. Thus one half of the graft will have a fibrous covering. It is dangerous to attempt to separate the deep surface of the rib extraperiosteally because of the danger of rupturing intercostal vessels before they are sufficiently exposed to be ligatured.

A subperiosteal graft, i.e., a graft without a fibrous covering, may be dovetailed into the diploic cavity at the margins of a defect after the tables of the skull have been prised open by suitable chiselling. Usually it is better to sew a graft in position by silk or wire sutures after corresponding holes have been drilled through its ends and through the skull. When holes are being made through the skull, the brain must be protected from the drill by means of a special pair of forceps (Fig. 162) that grip the bone between its two jaws, the upper of which is perforated to allow the drill to pass. When this instrument is not available a flat metal retractor may be slipped between the skull and the dura to protect the brain.

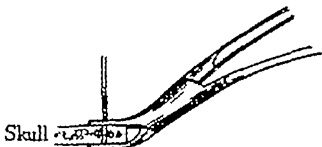


Fig. 162
Adson's drill forceps.

Grafts taken from the Tibia.—According to the area of bone required to repair a defect, a length of the superficial surface of the tibia from which it is proposed to take the graft is exposed through a straight or curved incision. If necessary manipulations are not to be impeded, a generous exposure of the tibial surface should be made. After the periosteum has been incised along the extreme anterior and antero-internal edges and separated centrally, longitudinal cuts are made in the bone with a curved hand-saw just within the line of the periosteal incision to a depth of 3 mm. Then periosteum and bone are cut transversely and a shaving of tibia 2 to 3 mm. thick is taken between the saw cuts by means of a wide chisel, the length of the graft depending on the method to be used for fixing it in position.

Fixation by the Bone-shelf Method.—After reflection of the scalp and separation of the dura, a decision has to be made along which axis of the defect the laying of the grafts will give the best cosmetic results. Usually grafts are best laid along the flatter of the two curves. Whether this is in the coronal or sagittal plane depends on which part of the skull the operation is to be done.

When the axis has been chosen, a sunken square or rectangle, as shown in Fig. 163, is made to receive the grafts by removing the outer table of the skull. Removal of the outer table may conveniently be done by means of a curved hand-saw and a chisel.

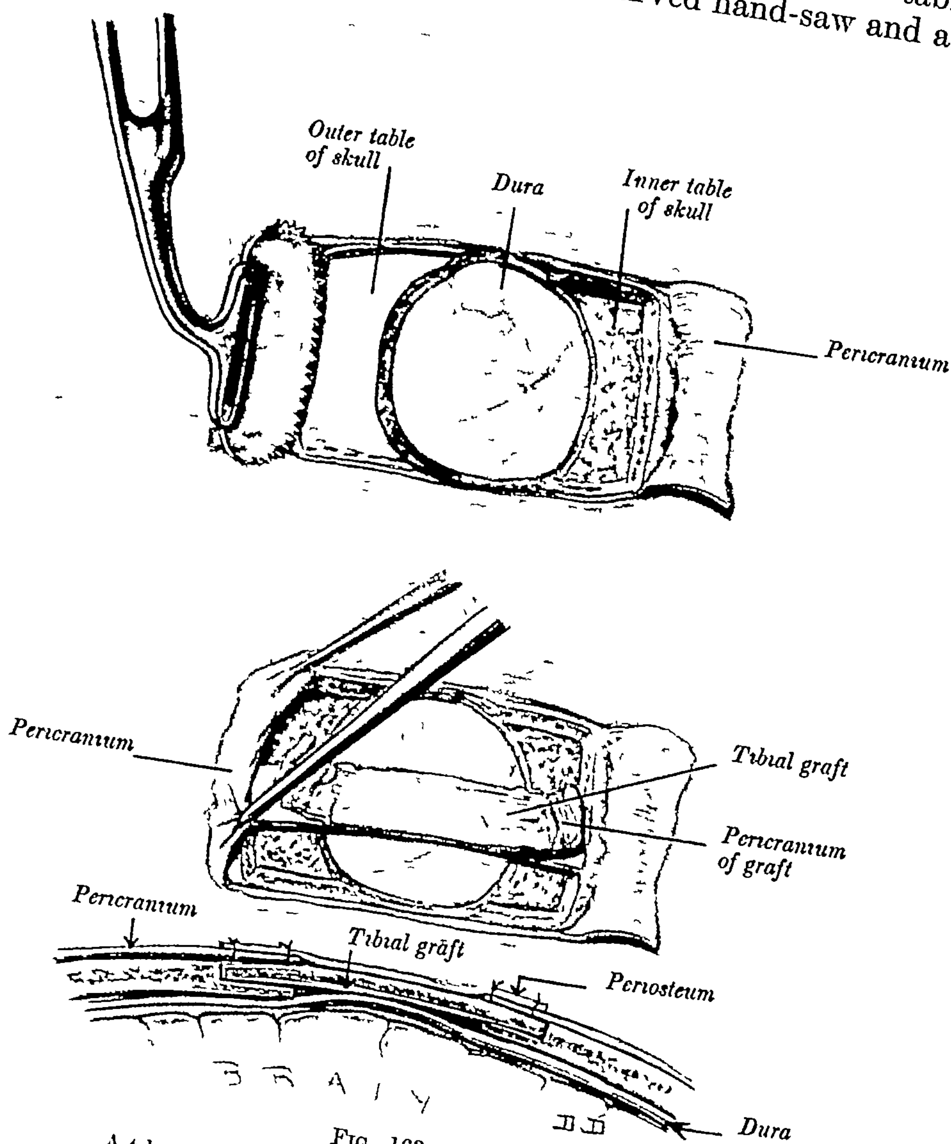


FIG 163

A tibial inlay graft for repair of a calvarial defect

The tibial grafts are then placed in position as an inlay and their covering periosteum sewn to that of the surrounding skull.

Fixation of Subpericranial Implantations.—This method has been used extensively by McGill and myself, and we find that its great advantage is its technical simplicity. Also, final consolidation of the graft by this method is as good as any other. Cosmetically it is satisfactory (Fig 164)

The bone grafts from the tibia are cut as described above, and of such length that they will overlap the edges of the defect by ¼ in at each extremity of the axis along which the grafts are to be placed The pericranium is then separated from the skull but not completely detached, and the periosteum at the ends of the grafts is lifted as a flap The denuded ends of the graft are then slipped beneath the curtain of pericranium and the grafts



(a)



(b)

FIG. 164

a A defect in the parietal bone b The same case after repair of the defect by means of a tibial graft placed between the bone and pericranium.

sewn into position as shown in the accompanying diagram (Fig 165). The scalp is sutured in the usual way drainage being unnecessary The patient may be allowed to get up in forty eight hours and after six weeks no protective covering on the head is necessary

Mowlem is now employing the method of grafting bone in the form of cancellous bone chips about a centimetre cube in size These bone chips form an easily contourable mass unite readily, and are excellent for those situations where good cosmetic results are of importance

REPAIR BY ALLOPLASTIC MATERIALS

The main advantage of utilising bone tissue from elsewhere in the patient's body in the repair of a cranial defect is that fibrous tissue reaction in the wound is minimised

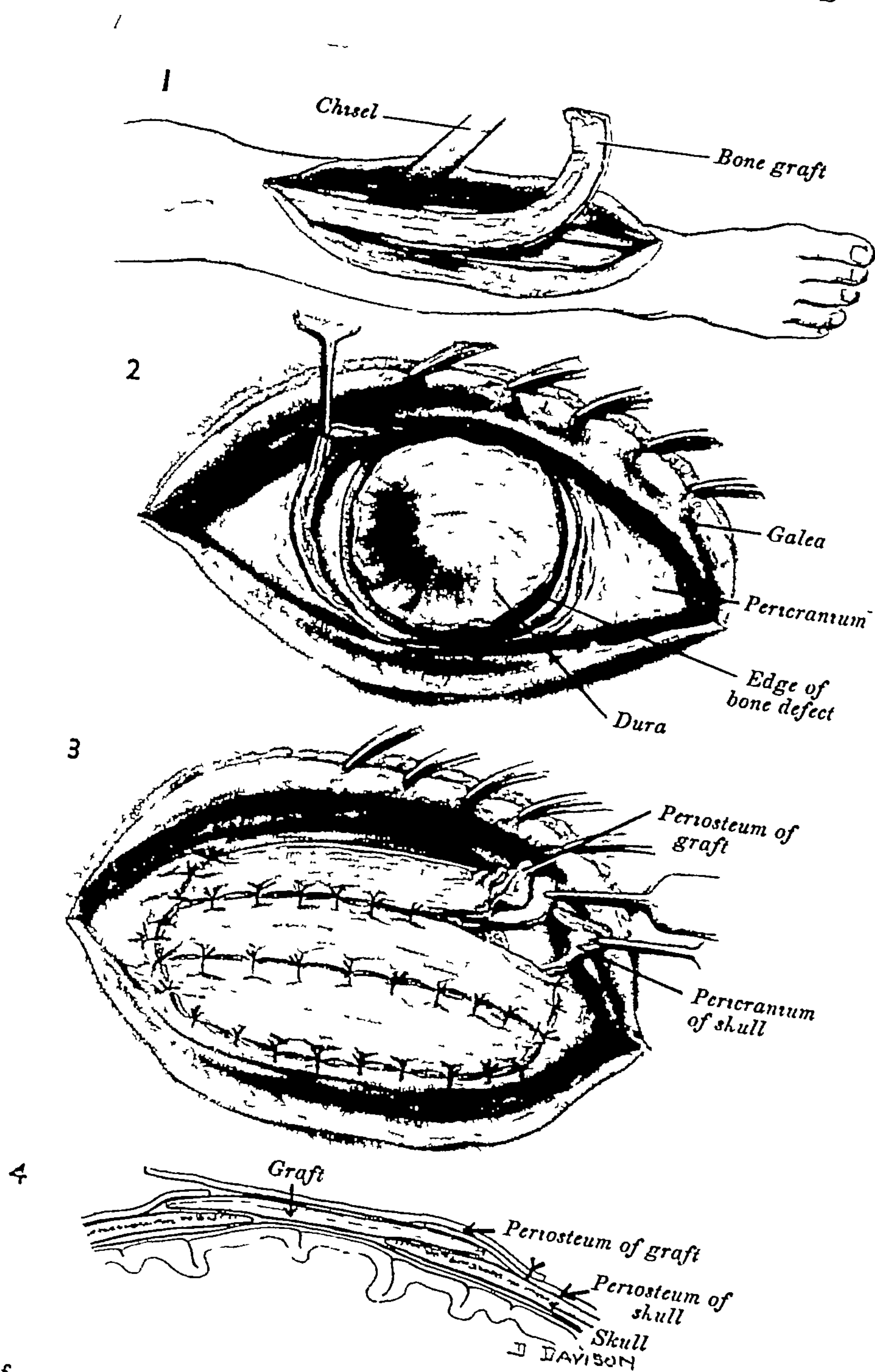


FIG 165

Repair of a calvarial defect by a tibial graft placed between the bone and pericranium

The disadvantages of homogenous grafts are —

(a) A more extensive operative procedure is necessary than when a ready prepared material is used

(b) Absorption of the graft sometimes occurs

(c) A good cosmetic result is difficult to obtain when the defect is situated about the orbit.

The two alloplastic materials now in common use are tantalum and acrylic resin My opinion is that alloplastic repair is the method of choice, and that tantalum is more useful than acrylic resin

Tantalum —The material tantalum is a bluish white easily malleable non rusting metal element occupying seventy third place in the periodic table Its main advantages are (i) that it is well tolerated by the scalp and skull and (ii) that it can be easily shaped and yet retain sufficient rigidity and strength adequately to serve its purpose of protecting the brain

One of its main disadvantages is that it is radio-opaque and so complicates further radiographic investigation should this become necessary as the result of epilepsy or abscess formation

Pre-operative Considerations —*The Scalp* —A tantalum graft must never be attempted unless the overlying scalp is healthy Thin scars will break down and latent infection become active Wounds that are not soundly healed should be opened and thoroughly explored Thin poorly vascularised scars should be excised and repaired if necessary by plastic measures As a broad rule, it is better to delay grafting for three months from the time of the original injury Chemotherapy is started on the day before operation

Shaping the Graft —By means of measurement and of comparison, either by radiography or through the skin the shape of the defect leaving ½ in overlap all round is marked out on a tantalum plate The graft is then cut out by means of heavy scissors and is shaped by the usual methods of metal beating Mallets of different weights and shapes made of hard wood are required with blocks of similar wood shaped so that large and small curves can be made on the graft For the making of grafts of complicated shapes a good deal of skill and patience is necessary The help of a professional metal beater may be necessary

The following method was devised by my colleague Mr G Hutchinson of the Dental Department of the Newcastle-upon Tyne General Hospital

Let us consider the shaping of a graft of complicated shape to fit a defect about the orbit The anterior half of the scalp is shaved the eyebrows are left untouched The patient is seated in a dental chair with his neck comfortably supported in

the head rest. He is instructed to close his eyes and to relax the muscles of the face and of the forehead. Leaving a margin of $\frac{1}{4}$ in., the defect is marked out with a line of methylene blue. The anterior half of the head and upper part of the face are smeared with castor oil. By means of plaster of Paris an impression is taken of the anterior half of the head and upper part of the face. It is made of such strength that it can be lifted away from the face, when set, without cracking. The outline of the defect is stained on the impression by a methylene blue line. This line is made permanent by pressure with a pointed metal instrument on the plaster, so that when the tantalum is being moulded a definite line of demarcation is seen on the external borders of the plate. The impression is lubricated and, by means of plaster of Paris, a cast of the patient's head is obtained. The indentation of the defect in the plaster cast is now filled in with dental wax and moulded so that both sides of the head are symmetrical. At this stage a reverse model is made in "stone-hard plaster" of the area to be repaired, allowing an overlap of at least 1 in. Then, using damp casting sand in a large metal ring, an impression is obtained from the stone-hard model and molten zinc is poured into the sand. When this cools a zinc replica of the defect is left. This is now covered with French chalk, and the base is embedded in sand in the metal ring and molten zinc is poured over the model. Normally, lead is used for this latter process, but it has been found that the careful use of zinc gives better results. In this way a die and counter die are obtained. A paper pattern of the defective area is now made, and the graft is cut to an approximate size from a tantalum plate and placed between the die and counter die. By means of a heavy press or by aid of a 6-lb. hammer, sufficient pressure can be exerted to press the graft into the desired shape. Final adjustments are made by fitting the prosthesis against the actual defect on the patient's head. A peripheral row of paired holes are made to enable the graft to be sutured to the bone, and the rest of the plate is perforated so that deep exudations can escape to the surface. Sterilisation is produced by boiling.

Operative Procedure.—If technical difficulties are to be minimised in the depth of the wound, the whole of the cranial area to be repaired must be freely uncovered by the raising of a suitable and sufficiently large skin flap. A graft may be placed on and stitched to the pericranium, or it may be slipped beneath the pericranium and stitched to this membrane. These procedures are known as the onlay method.

In all our cases we now use the inlay method and sew the graft to the bone by means of tantalum sutures. The shelf may

be cut either by chiselling alone or by a combination of chiselling and sawing, as shown in the diagram (Fig 163) It is essential for good results that the graft fits snugly It is wrong to try and anchor a badly fitting graft by tight suturing as the graft will almost certainly break from its sutures, slip, perforate the skin and have to be removed

Repeated post-operative aspirations of the wound are usually necessary, and must be carried out regularly to protect the skin flap from tension and stretch Prolonged tension under the skin flap will lead to thinning of the scar and to the danger of secondary breakdown As soon as the wound is healed the patient may be allowed to get up and about.[1-3]

Acrylic Resin.—The disadvantage of acrylic resin grafts is that two operations are necessary, with resulting danger of infection, also considerable time is consumed In acrylic grafts the wound has to be opened for the impression of the graft to be taken and then closed for a day or so until the graft has been made The wound is then reopened and the graft fitted into position

The advantage is that the snug fitting of a graft of complicated shape can be ensured A one-stage method for acrylic grafts is now being used, but has not yet been perfected [4 5]

SUMMARY

So far tantalum, in my opinion, has been the most suitable material for the grafting of defects in the skull However, I have retained the description of homogenous bone grafting because the long term results of tantalum grafting have not yet been proved I have, in the last year, removed two celluloid grafts that were used to repair defects in 1920 following the 1914 18 war

[1] Pudenz R. H "The Repair of Cranial Defects with Tantalum." *J A.M A.*, 1943 121, 478.

[2] Fulcher O H. "Tantalum as a Metallic Implant to repair Cranial Defects: a Preliminary Report." *J.A.M.A.*, 1943, 121, 931

[3] Lewin, W., Graham, M. P., and Northcroft, C. B. "Tantalum in the Repair of Traumatic Skull Defects. *Brit Jour Surg.*, July 1918, p 76.

[4] Small, J M., and Graham M P "Acrylic Resin for the Closure of Skull Defects: Preliminary Report *Brit Jour Surg.*, 35, 106.

[5] Oliver, L. C., and Blaine G "A New One-stage Method of Cranioplasty with Acrylic Resin.' *Med Press*, Sept 1948, p. 167

CHAPTER VI

OPEN OR COMPOUND WOUNDS OF THE HEAD

FIRST AID AND TRANSPORT

ROAD Accidents.—In road accidents shock and bleeding from the scalp are the only urgencies which a medical man is likely to encounter and have to treat when a patient has been injured about his head Other types of injury are either of such severity that they are beyond the aid of medical measures or can wait without deterioration for treatment until adequate surgical facilities are available Owing to shock, bleeding often stops spontaneously, but on those occasions when it does not, immediate action must be taken if excessive amounts of blood are not to be lost As it is only by chance that surgical material may be available, first-aid ambulance methods are usually necessary to still bleeding by the application of pressure. This may be done most conveniently by means of the two-handkerchief method (Fig. 166) The first handkerchief is put over the wound and then a second handkerchief or scarf folded into a triangle, with its base placed across the forehead, is tied in position by knotting its lateral tails below the external occipital protuberance By pulling on the apical tail, pressure can be applied over the first handkerchief and the bleeding stopped. Such bandaging, of course, must be done judiciously, otherwise loose fragments of bone may be driven inwards to compress or lacerate the brain

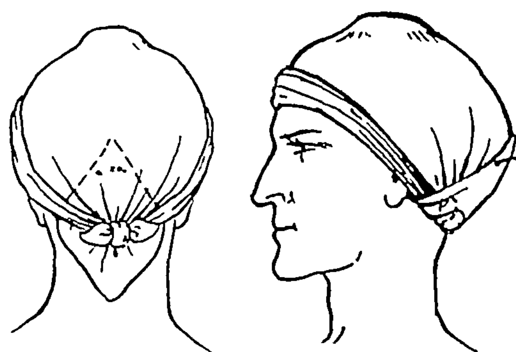


Fig 166
The two-handkerchief method of controlling bleeding in the scalp until surgical facilities are available

Air-raid Casualties.—During and after air raids little can be done for the unconscious with closed head injuries, and such patients should be removed to hospital as soon as possible.

Surgical outposts, as far as can be seen at the moment, are mainly concerned with open wounds, and here the main problems

are (1) the treatment of shock, pain, fear and restlessness, (2) the prevention of sepsis, and (3) decisions regarding transport.[1] A simple wound of the scalp or one associated with a clean linear fracture may be sutured at once if reasonable aseptic precautions can be taken, such as shaving of the scalp. A warning, however, is necessary not to overlook the possibility of a severe cerebral injury in the presence of a simple scalp wound. When a lacerated scalp is associated with a cerebral injury it is better to leave the wound open, apart possibly from a few tension sutures to control bleeding, because in such circumstances if a wound has been carefully sutured the surgeon at the receiving hospital may be tempted to leave it intact and not to explore as possibly he ought the depths of the wound. In depressed fractures it is wise always to be conservative and delay radical treatment until adequate surgical facilities are available, since the simple lifting of a depressed fragment may start uncontrollable bleeding. Also when the dura mater is opened, manipulations in the absence of perfect aseptic precautions will almost certainly lead to spreading encephalitis. Bleeding can usually be stopped by means of light pressure applied over a sterile dressing. On no account must the source of bleeding be sought deeper than the scalp, as this is almost certain to increase the hæmorrhage and disseminate infection. Bleeding from the edge of the scalp may be controlled by hæmostats or preferably by pulling the wound loosely together with sutures. To attempt to repair a complicated wound without adequate assistance and full surgical facilities shows a serious error of judgment.

Pain, fear and restlessness may necessitate the administration of small doses of morphia, but drugs are better withheld if there is no delay in transport. Adequate excision, wherever this may be done, is necessary for prevention of sepsis, and it is very wrong to regard chemotherapy as the panacea against infection. Shock is treated by rest and warmth; blood transfusion is necessary for hæmorrhage.

From the point of view of transport, the injured may be classified thus:—

(i) The moribund and those seriously ill.
(ii) The shocked and the exsanguinated.
(iii) The violently restless.
(iv) Those with minor injuries.

In times of stress it is permissible in severe cases to give precedence to those with the better prognosis and to keep the moribund

[1] Ministry of Health. Emergency Medical Service. "Memorandum on the Treatment of Head Injuries." E.M.S., Gen., 260.

behind, as the latter are likely to die before they reach the main hospital. In any case little can be done for them surgically when they have received severe intrinsic injuries to the brain.

Patients with minor injuries may be allowed to go home or may be sent for a short period to rest in neighbouring houses.

Whenever a wound has been sutured, the details of the exploratory findings must be stated quite clearly in the patient's notes, and it is essential to make it clear whether a satisfactory repair has been made or whether the wound must be reopened for more extensive excision at the main hospital. Moreover, it is always useful for a surgeon to know whether the dura mater has been lacerated or not, since tears of this membrane are in need of more urgent treatment than injuries confined to the scalp or bone As in closed head injuries, neurological details of the early phases of the injury are useful in guiding subsequent treatment.

The above suggestions and advice applied to the conditions of the 1939-45 war, and, no doubt, many of the same principles will obtain in future emergencies should they come early. We must now also consider what future developments there are likely to be. How, for example, shall we be equipped to treat injuries resulting from radio-activity; also, may not the artificial sunlight lamp be as important as the gas mask?

Battlefield Casualties.[1-8]—The treatment of acute head injuries on the battlefield depends, of course, on circumstances. It is conditioned by a complexity of interacting factors: by the state of warfare, whether the battlefield is static or mobile, and if mobile whether the Army is advancing or retreating; by terrain; by facilities for transport, by the number of casualties; by the medical personnel and by the immediate facilities for major operative procedure. The wide principles of medical and surgical treatment have already been laid down by the higher military authorities and the established organisations for the handling of battlefield casualties.

The object of this section is to guide the medical officer when conditions are such that he must use his own personal judgment to make a decision. At the outset it can be stated with confidence that a man suffering from concussion only, travels well on a

[1] Cairns, H "Gunshot Wounds of the Head in the Acute Stage" *Brit Med Jour*, 1944, Jan 8, p 37 *Ibid*, 1943, **1**, 313
[2] Cairns, H, Eden, K C, and Schorstein, J "Investigations concerning the Use of Penicillin in War Wounds" H M Stationery Office, London 1943
[3] Ascroft, P B *Brit Med Jour*, 1941, **1**, 739 *Lancet*, 1943, **2**, 211
[4] Eden, K C *Lancet*, 1943, **2**, 689
[5] Schorstein, J *Lancet*, 1944
[6] Sorgo, W *Zbl Neurochir*, 1942, **7**, 73
[7] Tonnis, W *Zbl Neurochir*, 1942, **6**, 113
[8] Money, R A, and Nelson, T Y "Experiences with Battle Wounds of the Head" *Ann Surg*, 1943, **118**, 1

stretcher or in an ambulance. Immediate or early transport is contraindicated when —

(a) The brain injury is overwhelming and the prognosis hopeless

(b) There are severe associated injuries

(c) Severe shock is present, as judged by the diastolic blood pressure or obvious clinical picture

(d) There has been considerable blood loss—in these cases a transfusion before removal is advisable

The experiences on modern battlefields so far have shown quite clearly that the sooner a head injury comes into the hands of a man skilled in traumatic head surgery the better are the results This raises the problem of whether a neurosurgical team should or should not function close to the fighting line. In France an invaluable neurosurgical team was captured in the early stages without being able to give continuing succour to the injured In Tunisia, where the troop movements were in the opposite direction the results of a neurosurgical team close to the front line fighting were truly remarkable The judgment of how close a special team should be to the active fighting is obviously one for the man in charge of the unit to make himself

After immediate transport problems have been settled the surgeon has to decide how much he himself can or is prepared to do with the facilities available to him It is on this aspect of the problem that the author feels he can give advice

Good surgery of the scalp is the basis of good results in head surgery If the scalp is properly shaved and the skin around the wound is cleaned and no more, then a good job of work has been done and the chances of a favourable result greatly enhanced It cannot be overstressed that correct preparation and cleaning of the scalp is an essential part of head surgery The golden rule of correct preparation is probably more often broken in head than in any other branch of surgery Apparently, to some people a head wound is either so simple that no particular care is needed or the injury so deep and so severe that whatever is done makes little difference. The chances of a good result are seriously jeopardised if the scalp is not properly prepared Moreover, *more than one wound may be present* Save in trivial wounds the whole head should be shaved Even if no immediate surgery is contemplated but there is a prospect of delay in transport the head should be shaved and gross contamination removed from the wound Sulphonamide powder and sterile dressings are then applied

When a surgeon has once embarked on the débridement of a head wound and finds that it is more complicated in the depths

than he had anticipated, he is quite justified in stopping. All that is necessary is for the wound to be packed with sulphonamide powder and for the case to be transferred to a special centre as soon as possible.

The one type of case which causes almost insuperable difficulties if complications necessitate further surgical treatment is that in which a primary closure has been made and the wound breaks down with resulting infection in the superficial tissues. The best results come from adequate primary débridement followed by sound primary healing of the scalp.

The previous section was written during the past war, and we are now in a position to see that few of the instructions laid down have to be altered The final lessons learned from the war were that —

(*a*) Early definitive surgery is essential if the best results are to be obtained in open wounds of the head

(*b*) Adequate resuscitation before operation not only saves lives but minimises morbidity rates.

(*c*) When first-aid measures only are available, local interference with the head wound should be the minimal that is compatible with the saving of life and the prevention of gross infection.

(*d*) Chemotherapy should be instituted as early as possible as a prophylactic measure.

General Considerations.—The principles of diagnosis and treatment in closed injuries of the head apply equally to open wounds of the vault A detailed neurological examination must therefore be made in all cases, however trivial a superficial wound may be, so as to determine whether or not an injury to the brain has occurred, and if so to localise it and to diagnose its nature. Without this information it is impossible to treat a case correctly, since local appearances in a wound may give no indication as to the damage done deeper in. For example, a linear fracture may have to be trepanned if there are neurological signs of an underlying extradural or subdural hæmorrhage.

Treatment, though primarily directed to excision and repair of wounds, is also designed to keep intracranial pressure within normal limits in the manner described in the previous chapter

Wounds of all types may be classified according to their depths,[1 2] thus —

(i) Wounds confined to the scalp.

(ii) Wounds associated with linear fractures of the skull.

[1] Cushing, H "Study of a Series of Wounds involving the Brain and its Enveloping Structures" *Brit Jour Surg*, 1917-18, **5**, 558

[2] Jefferson, G "War Wounds and Air-raid Casualties War Wounds of the Head—I" *Brit Med Jour*, 1939, **2**, 347

(iii) Compound depressed fractures with intact dura mater
(iv) Compound depressed fractures with torn dura mater
(v) Indriven fragments and retained missiles
(vi) Wounds opening into the mastoid or paranasal air sinuses.

It is important to know that the dura acts as a very efficient barrier to the spread of infection into the brain tissue or meningeal spaces In fact a classification of wounds could be made on the basis of dural integrity, since this is of the greatest prognostic value When the dura is intact a patient is very unlikely to die from infection if a wound is carefully excised On the other hand, when the dura is torn there is always grave danger of encephalitis and meningitis, however carefully or early débridement is carried out.

Pre-operative Considerations—A wound may be explored pre-operatively to determine its depth in order to get some kind of guidance as to the magnitude of operation that later may be necessary, and this is best done with a sterile finger, since probing with a metal instrument is more dangerous as regards dissemination of infection and less likely to give satisfactory information The main objection to pre-operative exploration is that troublesome bleeding may be started which is not easy to stop by simple means

Associated injuries are often present, and whether their treatment is more urgent than the head wound must be decided in each case Usually this decision is not difficult Although the ideal time to operate on a patient with a compound wound of the head is within twelve hours of the receipt of injury, many patients with multiple injuries may safely be left for eighteen or even twenty four hours from the point of view of infection

In any case it is most unwise to operate on a patient before he has recovered from primary shock, and certainly many cases are unnecessarily lost through failure to observe surgical first principles When in doubt about the ability of a patient to withstand an extensive operation on the head, it is better to wait than to be in a hurry In one case I delayed operation for two and a half days with gratifying results.

X-rays.—A pre-operative X ray examination of the skull must never be omitted as this gives direct evidence of what otherwise must often be conjectural It will show the extent of a bony injury the size of a depression, the number and exact site of indriven bony fragments and the presence or absence of radio opaque missiles, such as bullets or pieces of shrapnel In air raid casualties radio-opaque material is usually matted in the hair and therefore Harvey Jackson,[1] with much reason has

[1] Jackson, H Paper read at the meeting of the Neurosurgical Society Oxford, July 1941

suggested that radiographic examination is better done after the head has been shaved.

Choice of Anæsthesia.—Local anæsthesia is the method of choice in the treatment of a compound wound of the head. It is safer than other methods from the patient's point of view, and as a surgeon becomes accustomed to its use he will soon realise how much easier it makes the actual operating. By this means intracranial pressure is minimised and troublesome venous oozing avoided. Moreover, when the dura mater is torn a general anæsthetic is dangerous, because it invariably causes a rise of some degree in intracranial pressure, which may initiate or increase an existing cerebral hernia

Novocaine should not be injected into or near the edges of a wound but some distance away, in the form of a ring to enclose the proposed operative field This procedure not only keeps the anæsthetic clear of damaged and infected tissues but also allows easy extension of a wound if this is found necessary.

General anæsthesia[1] is indicated when a patient is restless and unco-operative and when the scalp is so extensively lacerated that it is impossible to shave it without causing severe pain (see Chapter IV) It may be necessary to use a general anæsthetic if other injuries are to be treated at the same time as the head wound. When a patient is fully conscious and has recovered from shock, a general anæsthetic may be given, provided that the dura mater is intact.

THE SCALP

Applied Anatomy.—The calvarium or vault of the skull has five coverings and each one is surgically important. From within outwards these are :—

(i) The pericranium.
(ii) Loose areolar tissue.
(iii) Galea aponeurotica.
(iv) Superficial fascia
(v) Skin.

The pericranium (Fig 167) is firmly attached to suture lines but readily separable elsewhere, and therefore hæmorrhages in the subpericranial layer remain confined within the boundaries of a single bone area. Examples of this type of bleeding occur in birth injuries when so-called cephalhæmatomata are to be seen standing up like mounds on the head, in the region of the parietal bones.

The Layer of Loose Areolar Tissue.—The looseness of the attachment of the areolar tissue, both to the galea and to the

[1] Brennan, H J "Gas-oxygen and Cerebral Congestion" *Lancet*, Feb 1938, 1, 315

pericranium, explains why the skin can move freely over the surface of the skull This is of great surgical importance, since it allows closure of a wound even after considerable areas of tissue have been lost. Another point of surgical importance is that infection can spread rapidly with little hindrance along the areolar layer If, therefore, serious complications are to be avoided, radical and early drainage of suppurative processes in the subgaleal space is essential

The galea aponeurotica or the tendon of the occipito frontalis muscle is attached directly or indirectly to the whole circumference of the base of the calvarium thus forming a fibrous tissue envelope for the skull A wound involving only the skin does not gape, whereas when the galea has also been incised gaping does occur The galea is of particular surgical value in the repair of extensive wounds of the scalp because it is tough and may be

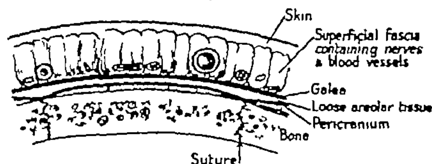


FIG. 167
Cross-section of the scalp.

sewn together with buried sutures, which allows skin sutures to be removed in a few days' time. This minimises the dangerous possibility of stitch abscesses and also to a large extent eliminates disfiguring scars

The Superficial Fascia or Subcutaneous Tissue—This tissue binds the skin proper firmly to the underlying galea and therefore no movement is possible between the more superficial layers of the scalp Also it is in the layer of superficial fascia that the main nerves and blood vessels are found, and owing to the fibrous nature of the tissue normal retraction and contraction of severed blood vessels is prevented, so that profuse bleeding occurs when the scalp is cut

The skin is so richly supplied with blood vessels that even in a small wound of the scalp a patient may lose a considerable amount of blood in a very short time In view of this, therefore, very great care must be taken when operating on the head to have the blood vessels under control by digital compression before cutting them The advantage of a rich blood supply is that wounds

heal well and that flaps of tissue with a small base will live. The richness of the scalp's nerve supply means that cuts of the scalp and operations on the scalp cause considerable shock if the skin incision is not blocked with local anæsthesia.

The Nerves of the Scalp (Fig. 168).—The trigeminal nerve

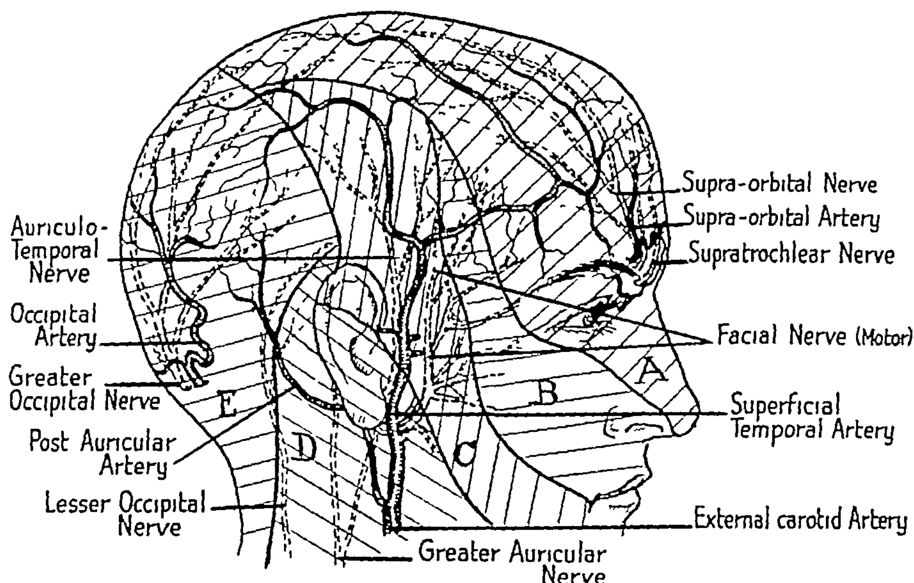


FIG 168

The blood vessels and nerve supply of the scalp

A, Area supplied by ophthalmic division of the trigeminus, B, area supplied by the second division of the trigeminus, C, area supplied by the third division of the trigeminus, D, area supplied by the anterior divisions of the upper cervical plexus, E, area supplied by the posterior divisions of the upper cervical plexus

supplies the anterior two-thirds of the scalp and the cervical plexus the posterior third.

(*a*) *Branches of the Trigeminal Nerve*—

Division 1.— (i) Supra-trochlear.
(ii) Supra-orbital
Division 2.—(iii) Zygomatico-temporal.
Division 3.—(iv) Auriculo-temporal.

(*b*) *Branches of the Upper Cervical Plexus*—

(i) The great auricular.
(ii) The lesser occipital.
(iii) The great occipital.

The Blood Vessels of the Scalp.—The blood vessels on each side of the scalp freely anastomose. Except in the region of the forehead, however, anastomosis is poor across the middle line. Where possible incisions in the midline on the top and at the back of the head should be avoided. Also, as the arteries of the

scalp run from below upwards, incisions in the scalp should be made vertically and not transversely Skin flaps should be turned downwards and not upwards

The arteries of the scalp originate from

(a) Internal Carotid Artery { Frontal, Supra-orbital }

(b) External Carotid Artery { Superficial Temporal, Posterior Auricular, Occipital }

Veins.—The frontal and supra-orbital veins drain via the ophthalmic veins into the cavernous sinus

The temporal and posterior auricular veins drain into the internal jugular veins

The occipital veins drain into —

(a) The deep cervical veins
(b) The vertebral veins

The veins of the scalp are freely connected with the parasagittal lateral and cavernous venous sinuses through so called emissary veins Not only, therefore does bleeding occur from the bone when the flap is lifted because of these communicating channels but also infection from the scalp can easily gain access thereby into the intracranial cavity by means of thrombophlebitic processes

Lymphatics of Scalp —

(i) The occipital region drains into glands on the insertion of trapezius muscle at the back of the head
(ii) The parietal region drains into glands behind the ear over the insertion of sternomastoid muscle
(iii) The anterior region of the scalp drains into the glands in front of the ear over the parotid gland

SURGERY OF THE SCALP

Preparation of the Scalp —Unless a wound is small and obviously superficial the whole scalp should be shaved and the skin cleaned in the usual way Local shaving is to be condemned since the adjacent hair becomes matted with blood and acts as a breeding ground for bacteria. Moreover, difficulties may arise at the time of the operation if the operative field has to be enlarged

In air raid casualties shaving of the head has proved to be an extremely difficult and lengthy manœuvre because grit and

dirt blown into the hair and skin by the explosion blunt the razor edge after a few sweeps across the scalp. The method I have found most useful is to cut the hair short with scissors, comb it,



(a)





(b)

FIG 169

A scraping injury

This man was dragged by the legs face downwards with his face bumping along a road down a coal mine

(a) Before operation

(b) After operation, note how little skin was sacrificed in the débridement Later, further plastic measures will be carried out and the man fitted with a glass eye

then to scrub it with a sterile nail-brush and soap and water and finally to shave it. Botterell has shown that a safety-razor blade gripped in the jaws of an artery forceps is an excellent way of always having a sharp and efficient tool at hand. Prepara-

tion of a head is best done in a bathroom where the patient can be thoroughly washed with warm water all over the body, because large quantities of dirt often collect in the axillæ and groins and cause discomfort

Infection of the scalp always leads to prolonged suppuration Therefore if valuable time is not to be wasted, both for the patient and for the hospital staff concerned every wound however trivial must be carefully cleaned, trimmed and accurately sutured (Fig 170) The exact details of an excision naturally will depend on the size, shape raggedness or degree of contamination of a wound The main principles to be observed in débridement are that edges of skin should be cut away sparingly with a sharp scalpel and that all foreign bodies should be removed from the depth of the wound



Fig. 170

Cellulitis of the scalp is often the result of an inadequate excision of a simple wound.

Plastic Operations for Closure of Defects in the Scalp—Though the skin is firmly attached to the galea, the galea moves freely over the pericranium, and thus, with suitably placed incisions, the edges of a wound may be mobilised and freely retracted if a wider exposure of deeper tissues is found necessary On those occasions when tissue has been lost forcible apposition of the skin by mattress sutures is useless, because the margins of the flap will certainly slough and the wound break down The maximum tension permissible in a wound difficult to close can be judged by buried sutures, and it may be accepted as a working rule that if the skin can be brought together by fine silk sutures passed through the galea, tension will not be too great to prevent healing By suitable incisions and sliding of the scalp a wound may be drawn together even if there has been considerable loss of tissue The main methods are as follows —

(a) *Elliptical Excisions*—The conversion of a defect into a long ellipse is a most useful method of effecting closure of a wound which is not very ragged and in which loss of tissue is not great A modification of this method is to prolong the extremities of the incision in the form of an S (Fig 171).

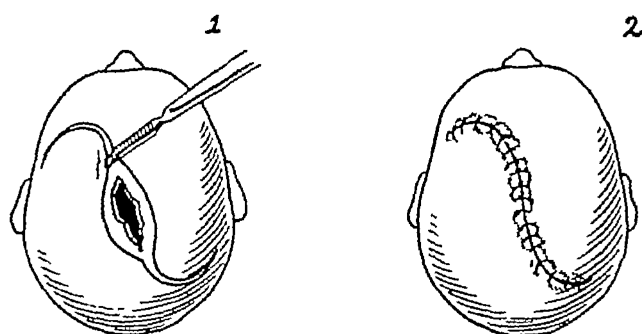


Fig 171

The S-shaped incision showing closure

(a) (b)

(c)

Fig 172

Triradiate incision of Cushing.

(a) Principles of curved tripod indicated by arrows.
(b) Curved tripod suture
(c) Method of closure of curved tripod

(*b*) *Triradiate Incision of Cushing*.—In this method three incisions from the extremities of a ragged defect are carried out into normal scalp as shown in Fig 172. That these plastic incisions should be correctly shaped and sutured has been stressed by Gillies.[1]

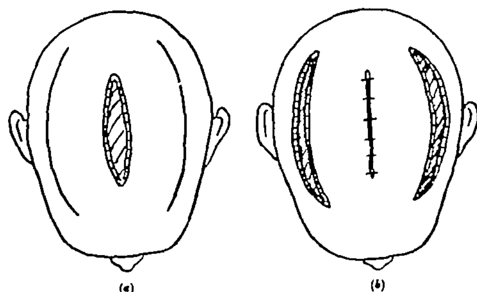


Fig 173

Relaxation incisions.

(*a*) Note these are longer than wound.
(*b*) Suture of wound. Relaxation incisions unsutured



Fig. 174

The quadrilateral flap.

Suppose there is a defect or unhealthy scar in the above position. A flap is marked out A, B, C, D. The defect is then cut out by removing the triangle C, E, D; D, E, B, A is then swung forward and the flap is sutured in the new position.

[1] Gillies H "Note on Scalp Closure" *Lancet* 2nd September 1944 p. 310

(*c*) *Relaxation Incisions.*—These are useful for the prevention of tension in linear wounds This method is an excellent one for suitable cases. It is important, however, to make the relaxation incisions longer than the wound itself (Fig. 173)

(*d*) *The Quadrilateral Flap*—This is the method that was most utilised by the author in his early work and still remains an excellent method in certain types of case (Fig. 174).

(*e*) *The Sliding Rotational Flap of Gillies.*—This is probably the most valuable plastic manœuvre that has yet been described. Most defects, whatever their shape or position, given that not too much tissue has been lost,

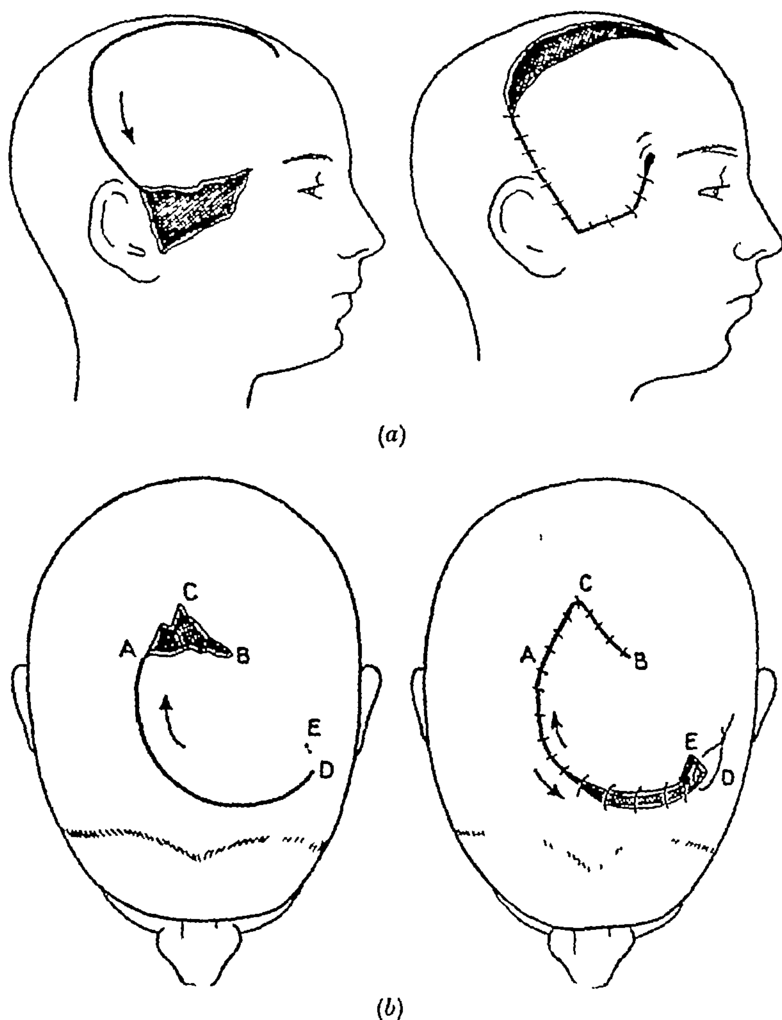


FIG 175

The rotational flap of Gillies.

(*a*) For complicated wounds in the temporal region
(*b*) For tissue loss on the vertex

can be satisfactorily repaired by this method In large defects, after the flap has been swung and the defect



FIG. 176

When a large area of scalp has been destroyed and the underlying bone removed, a skin flap from a neighbouring region can be swung to repair the defect The pericranium in the denuded area is left intact when the skin is raised to allow immediate repair by a Thiersch graft In thi case the bone defect lay in the occipital region.



FIG 177

A Thiersch skin graft is valuable in the repair of a wound where there has been so great a loss of scalp tissue that the wound cannot be drawn together even by plastic manœuvre

satisfactorily covered, it may be impossible to appose the skin edges at the back end of the wound and a

bare area will be left. This area can be covered immediately by a Thiersch skin graft (Fig. 176).

(*f*) *Skin Grafting*—In those cases when it is impossible to close the skin completely in spite of the employment of plastic incisions, the exposed part of the wound should be covered immediately with a Thiersch skin graft taken from the thigh.

Electrical Burns of the Scalp.—In the past few years, through the courtesy of Professor J. C. Spence of the Pediatric Department of the University of Durham, I have had referred to me for surgical treatment four cases of electrical burnings of the scalp.



Fig. 178

If the morbidities of sloughing infection and osteomyelitis resulting from electrical burns are to be avoided, the affected area of scalp must be cleanly and boldly excised through healthy tissue. Timid and inadequate surgery leads inevitably to failure.

On this experience I can advise without hesitation that a wide and bold excision of every severe electrical burn of the scalp should be made immediately after shock has been treated. The defect is then repaired by means of one of the plastic operations described above. It is essential that no dead scalp tissue be left behind, otherwise the wound will break down and become septic. It is not necessary to remove any underlying bone, as all that will happen to this tissue is an aseptic necrosis that probably will give rise to no trouble in the future. Also, possible brain damage can be best left to the care of natural processes. In those cases where adequate excisions and repairs are not made, infection almost certainly will occur and lead to a chronic and intractable osteomyelitis, which is very difficult to cure.

THE SKULL

Instruments used for the excision of skin are discarded as they are no longer sterile and the operation field is shut off with tetra cloths clamped into position with Michel clips As all open wounds are actually or potentially infected, it is important to know the type of organism concerned as a guide for future therapy, and so a sterile lintine swab is gently rubbed into the wound and sent for immediate bacteriological examination

Pericranium —An encircling incision is made through the

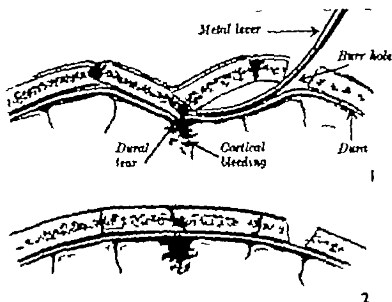


Fig. 170
A method of raising an indentation.
1 Shows a metal lever passed into position through a burr hole made at the edge of the depression.
2 By this method a contusion of the cortex may escape recognition.

pericranium ½ in or more if necessary, beyond the periphery of the depressed bony fragments and the lacerated and presumably contaminated membrane is then swept from the bone in sheets with a sharp rongeur and removed.

Linear Fractures —An uncontaminated linear fracture without displacement may be left undisturbed if there is no neurological evidence of underlying injury to the brain. On the other hand, when soiling or displacement has occurred it is safer to excise the fracture line by converting it into a narrow gutter with nibbling forceps, after a small burr hole has been sunk at one of its ends

Indentations —In funnel shaped depressions, though the bone is broken the fragments are not always free Occasionally

they may swing inwards on a hinge of intact pericranium and so may be levered into position and left *in situ* if the wound has not been soiled Elevation in these cases is most easily accomplished by sinking a burr hole through sound bone at the periphery of the indentation through which a curved dissector can be introduced and used as a lever of the first order, the fulcrum being the distal edge of the burr hole (Fig 179). When an indentation is deep or its apex spiculated as shown by radiography, it is wiser, after the bone has been raised, to cut a small gutter from the operative burr hole to the centre of the fracture so that the dura may be examined and repaired if torn (Fig. 180). This is important, since an unrepaired tear of the dura or a piece of bone left sticking into the cortex is apt to lead to epilepsy

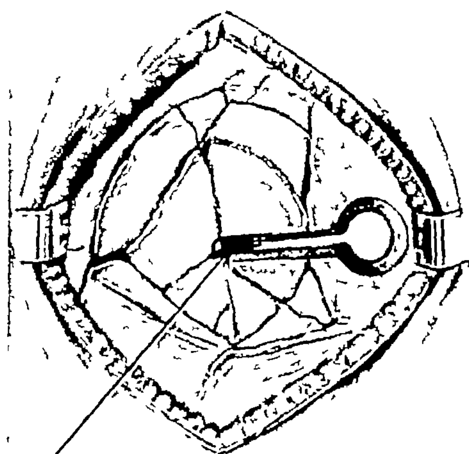


Fig. 180

Exploration of the dura immediately beneath the point of an indentation

Depressed and Interlocked Fragments.—In compound, comminuted and depressed fractures of the skull it is safer to remove all loose fragments of bone, as this helps to ensure primary healing Also, loose fragments lead to prolonged suppuration should infection supervene, and have to be removed before healing will take place. No qualms need be entertained on the score of free removal of bone, since repair of the skull at a later date is a relatively simple matter. Often in depressed fractures the dura is torn, and therefore when fragments of bone are interlocked great care must be taken not to lacerate the dura further with surgical instruments as an effort is made to disentangle the broken bone. When a depression is of small dimensions it is often useful to define intact dura from a burr hole sunk through sound bone at the periphery of the wound before attempting to elevate the bony fragments. This allows the fracture to be approached from the periphery and the plane of the dura always to be kept in sight. In extensive depressions interlocked fragments are best removed by block resection. This is done by burring four holes through the skull at convenient points around the fracture and by lifting out broken fragments in a block after the burr holes have been connected with linear cuts made either with a de Vilbiss forceps or with a Gigli saw (Fig 181).

There are, of course, exceptions to radical removal of bony fragments, and each case will have to be judged on its merits. For example, when a whole bone has been loosened but not badly displaced, it may be left in position, since the risk of its becoming infected is counterbalanced by an extensive and unsightly post-operative defect.

Fractures involving the Paranasal Air Sinuses[1,2]—A discharge

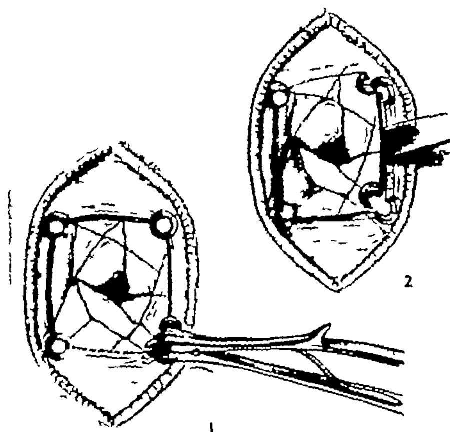


Fig. 181

Removal of depressed bony fragments by means of block resection. The instrument used in this case for the linear bone cut is a de Vilbiss forceps.

of cerebrospinal fluid from the nostrils means that a fistula has developed between the subarachnoid space and a paranasal air sinus or the nasal cavity. This condition is known as cerebrospinal rhinorrhœa and may present itself immediately after an injury or may not develop until some time later.

Immediate Cerebrospinal Rhinorrhœa.—In severe basal injuries a discharge of cerebrospinal fluid from the nose is by no means

[1] Cairns, H. "Injuries of the Frontal and Ethmoidal Sinuses." *Proc. Roy. Soc. Med.*, 1942, 35, 809.

[2] Calvert, C. A., and Cairns, H. "Discussion on Injuries of Frontal and Ethmoidal Sinuses." *Proc. Roy. Soc. Med.*, 1942, 35, 805.

[illegible] and would be far more frequently recognised if it were not masked by concomitant bleeding. Also, patients receiving the type of injury which causes this complication often die of cerebral injury before infection has time to develop. On those occasions



(a)



(b)

Fig. 182

A slicing injury.

(a) Before operation.

(b) Ten days after operation. The displaced bone was washed, rotated and then fitted back into position. Healing was by first intention, and was no doubt aided by the giving of penicillin both locally and systemically.

when patients do not succumb from laceration of the brain, meningitis may develop at any time after the injury, and no patient is free from this danger until extravasated blood has been absorbed and the fistula closed. Such healing may take many months.

1. Fractures of the Ethmoid Bone.—Theoretically, a compound tear of the basal meninges owing to fracture of the ethmoid

bone should be explored and repaired at once if serious infection is to be prevented Few surgeons, however, choose to operate immediately in these cases (1) because many of the patients are severely concussed and (2) because it is believed that many cerebrospinal fluid fistulæ heal spontaneously without meningitis supervening Moreover, it is known that the bony injury is usually extensive and lacerations of the dura ragged, so that repair, apart from the magnitude of the approach, is an operation of considerable proportions and not the simple obliteration of a single and isolated fistula.

On the other hand Cone[1] believes that many lives are to be saved by immediate operation on all compound tears of the basal dura. He argues that shock may be treated while a patient is on the operating table, and that it is illogical to leave repairs to the chance of fortune when it can be done deliberately under direct vision

The absolute indications for immediate operative repair are —

(i) Pre-existing infection in the affected air sinus
(ii) The onset of sinusitis, osteomyelitis or meningitis
(iii) Radiographic evidence of extensive displacement of fractured surfaces

As far as I can see the problem, operation is necessary in those cases when recovery from cerebral injury seems certain, but doubtful when the degree of concussion is such that the life of the patient is in the balance

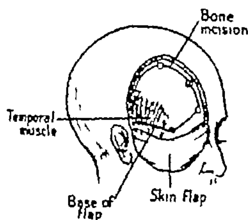


FIG 163
The unilateral approach to the floor of the anterior fossa.

Once a decision to operate has been made, the best mode of approach has then to be considered Usually radiographic studies of the fracture will determine from which side of the skull a fistula should be exposed, and stereoscopic views will be found particularly useful in making a decision on this point When radiography shows that the floor of the anterior fossa on both sides has been fractured and bilateral displacement has occurred, a bilateral exposure will be necessary

The details of the approach to the anterior fossa are as follows —

(a) *Unilateral Approach*—The scalp is incised along a line which starts at the glabella and then runs vertically in the

[1] Cone W V Personal communication.

midline of the forehead to within the hairline and curves downwards and outwards into the temporal fossa to end just above the upper border of the zygoma 1 in. in front of the external auditory meatus. This skin flap is raised and is reflected downwards. Burr holes are then sunk through the bone at points shown in the accompanying diagram and united by saw cuts (Fig. 183), care being taken not to open the frontal air sinus. The

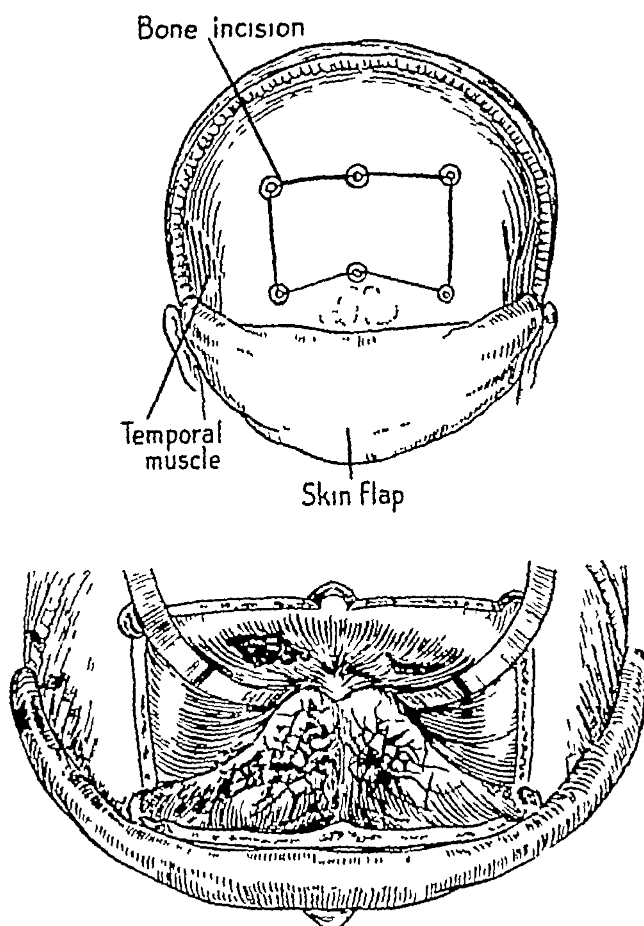


FIG 184
Exposure of a bilateral fracture of the ethmoid bone through a centrally placed bone flap

danger in opening this, of course, is the introduction of unnecessary infection. On the other hand, a bone flap must be as low as possible, otherwise access to the base of the skull may be so difficult that a posteriorly placed dural tear cannot be exposed. Loose fragments of bone should be removed freely and the openings into the ethmoid air cells covered with a sheet of tissue taken from the fascia lata of the thigh. Dural tears are best repaired by suturing, but when this is impossible a fascial or amniotic graft should be sewn across the dural defect

Drainage of the wound is not advisable if it can be left reasonably dry. At the end of the operation the bone flap is accurately replaced and anchored in position by stitching its periosteal covering to that of the surrounding skull. Finally, the skin is sewn together by two layers of sutures.

(*b*) *Bilateral Approach*.—The accompanying diagram will illustrate the line of the skin incision and the area of bone to be resected (Fig. 184).

One of the difficulties of this operation is experienced as the bone is lifted: veins entering the superior surface of the sagittal sinus are torn and give rise to troublesome bleeding if muscle grafts have not been previously prepared to control it. As in the above operation, loose bony fragments are removed, fascial grafts are placed across the fractured ethmoid bone and the dura repaired according to circumstances either by suturing or by grafts. The absence of a muscle or fascial attachment to the resected bone fragment makes no difference to its ability to consolidate with the rest of the skull when replaced in position. Fixation of the bone may be done by periosteal sutures or by sutures passed through drill holes made at corresponding and suitable sites in the skull and bone flap. Again the skin is sutured together in layers.

2. **Fracture involving the Frontal Air Sinus** (Fig. 185).—The frontal air sinus is, for obvious reasons, particularly vulnerable to injury. Even though the skin of the forehead is not broken the mucous membrane of the sinus is often torn and bleeding occurs into its cavity. The danger of this happening is that the clot may become infected and lead to sinusitis or to more serious complications such as extradural abscess. Fractures of the frontal air sinus are particularly difficult surgical problems when the overlying skin is broken, because adequate débridement of a wound necessitates complete removal of the many ramifications of the lining mucous membrane if the development of an unsightly external fistula is to be prevented. Fractures of the posterior wall often lead to fatal complications, because the dura is apt to be torn and infection and air allowed access to the meningeal spaces.

As in fractures of the ethmoid bone, decisions regarding the necessity and extent of treatment are largely guided by detailed radiographic studies.

It is unwise to lateralise the fistula of a cerebrospinal rhinorrhœa by the nostril from which fluid is escaping, as will be seen from the account of the following case.

The schoolboy son of a doctor, while riding on his pedal cycle to work in the fields, crashed head-on into a telegraph pole and was concussed. He very quickly recovered consciousness and

no residual signs of local damage to his brain were discovered. A few days later it was noticed that cerebrospinal fluid was leaking from his nose At this stage he was transferred to my care. Cerebrospinal fluid was found to be trickling from the left nostril but none from the right. Radiographs of the skull, however,



Fig 185
A fracture of the frontal air sinus is often best demonstrated by a routine "sinus shoot"

showed that a fracture of the frontal bone, which extended on to the base, was confined to the right side. Also, the frontal air sinus was opaque. This opacity, presumably, was due to bleeding, and as the extravasated blood was absorbed and the fundibular canal reopened, cerebrospinal fluid began to escape from the right nostril, thus declaring the true side of the fistula. Leakage from the left nostril was due to cerebrospinal fluid crossing the middle line from the right frontal air sinus to the left.

Frontal sinus injuries are best considered in two groups (1) closed, *i.e.*, when the skin of the forehead is intact, and (2) compound, *i.e.*, when the overlying scalp is broken [1,2]



FIG 186.

Displacement of the posterior wall of the frontal sinus of this degree is an indication for surgical exploration.

1 *When the Overlying Scalp is Intact* —When the skin overlying a damaged sinus is intact immediate operation need not be undertaken if there is no evidence that the dura has been

[1] Coleman, C C "Fracture of the Skull involving the Paranasal Sinus and Mastoids." *J A M A.*, 1937 109, 1613.

[2] Lockett W H "Air in the Ventricles of the Brain following Fracture of the Skull." *Surg Gyn & Obst.*, 191 24, 362.





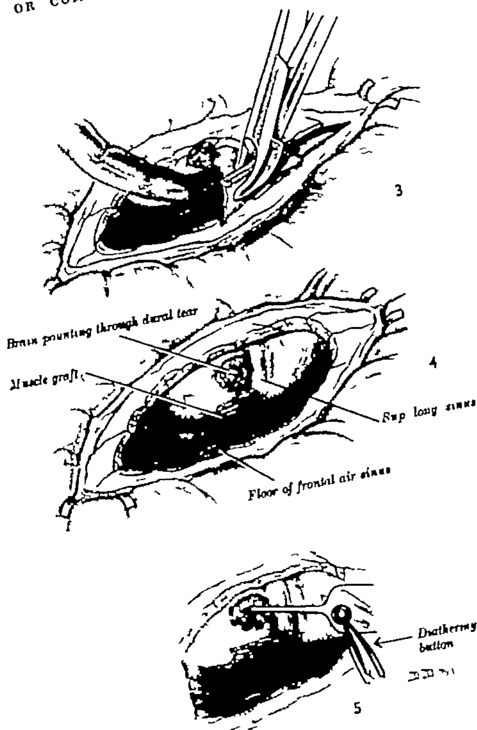


Fig 188

Excision of compound fracture of the frontal air sinus in which the dura has been lacerated.

3. Removal of loose bony fragments of the posterior wall of the sinus.
4. Control of bleeding from the sagittal sinus by means of a muscle graft.
5. Diathermy coagulation of a ruptured cortical vessel

torn and if the sinus was not diseased pre-operatively. Occasionally a surgical emphysema of the face and forehead develops, but as this usually resolves spontaneously and rarely becomes infected, incisions for its relief are unnecessary, and in fact dangerous. If infection should occur, immediate operation is necessary, and this should be done with the co-operation of an experienced rhinologist. On those occasions when radiography shows extreme displacement of the posterior wall (Fig. 186), or when a cerebrospinal rhinorrhœa has developed, exposure of the fracture and repair of the dura is necessary through a unilateral bone flap placed just above the damaged sinus.

2 *When the Overlying Skin has been Broken.*—When the scalp is broken and a clean linear fracture of the anterior wall of the frontal sinus has been found with no displacement, it is necessary only to excise the skin and remove damaged periosteum. The fracture line itself may be safely left undisturbed. When bony fragments from the anterior wall have been driven into the sinus they must all be carefully picked out and free drainage obtained by enlarging the infundibular opening into the nose. External drainage should be avoided at all costs as this will certainly lead to the formation of a persistent fistula. Though great care must be taken to remove all loose bony fragments, every effort must be made to conserve the supra-orbital ridge, otherwise unsightly deformity will result. In those cases where obvious contamination of the cavity of the sinus has occurred, prolonged suppuration will result unless the lining mucosa is completely removed; this part of the operation being carried out by a combination of suction and dissection under good illumination. Without good lighting facilities and suction, operations on any sinus are apt to be imperfect.

In compound fractures of the posterior wall of the frontal sinus, whether there is a discharge of cerebrospinal fluid into the wound or not, the dura should always be explored, so that tears may be repaired immediately if this proves necessary (Figs. 187 and 188). In such cases the superficial part of the wound is excised and the mucous membrane removed before the posterior wall is disturbed so as to make the operation as aseptic as possible. Dural repairs are carried out as described previously. At the end of the operation a rubber tube passed from the operative field through the infundibulum into the nose is allowed to drain the wound intranasally for forty-eight hours, after which it is removed.

Delayed Cerebrospinal Rhinorrhœa.—Leakage of cerebrospinal fluid from the nose may occur (1) a few days after injury, (2) during the period of convalescence or (3) at any time up to a period of months or even years.

In the first two types the fistula is probably produced at the moment of injury, but remains blocked for a time by blood clot, by a mucous plug or by an air bubble. Then owing to some simple change in the mechanical conditions, such as alteration in the position of the head, a rise in intracranial pressure or absorption of blood, the fistula opens and cerebrospinal fluid escapes. Why leakages should occur after months or years is much more difficult to understand. Some, possibly, as a result of the pull of cicatrisation of healing processes, are due to widening of a pre-existing but exceedingly narrow fistula which has passed unnoticed. Most probably they are to be accounted for by an aseptic necrosis affecting a local area of the cribriform plate or roof of the frontal air sinus. This process of osteitis dissecans may be the result of ischæmia of the bone, consequent on rupture of its nutrient vessels as the dura (endosteum) is stripped at the time of the accident. If for any reason the dura and mucous membrane covering the affected area of bone on either side become involved in the processes of necrosis, a fistula will result.

Diagnosis is simple. A colourless fluid trickles from the nose in drops and this discharge may be made more profuse by straining or by bending the head forward. Occasionally a leakage of cerebrospinal fluid may be confused with a watery discharge, either from the lachrymal gland or mucous membrane of the nose. Since cerebrospinal fluid is the only fluid which contains sugar, a chemical analysis in these cases is all that is necessary to distinguish cerebrospinal fluid from secretions arising within the nose.

In these delayed cases the side of the nose from which the discharge comes may lateralise the fistula, and intranasal inspection will show whether it is situated anteriorly or posteriorly. When the fluid is seen to be trickling from above the middle turbinate, the fistula passes through the ethmoid or sphenoidal air cells, but when the fluid drips from beneath the anterior end of the middle turbinate the fistula is more anteriorly placed and passes through the frontal sinus.

If necessary, methylene blue injected into the ventricles will definitely determine the presence of a fistula, since in such cases the discharge from the nose will be coloured blue.

Treatment.—Treatment consists of repair of the fistula through a unilateral frontal approach as described previously (Fig. 189). Recently Julian Taylor[1] has shown that an intradural approach to a chronic fistula has advantages over the better known extradural approach, by this means manipulations in a possible septic field

[1] Taylor, J. Paper read by Mr Eden at the meeting of the Neurosurgical Society, Oxford, July 1941.

are avoided and a view across the middle line is easily obtained if this proves necessary.

Craniomastoid Injuries.—As noted in a previous chapter, bleeding from the ear does not necessarily mean that the dura mater has been torn; in fact it does not necessarily indicate a

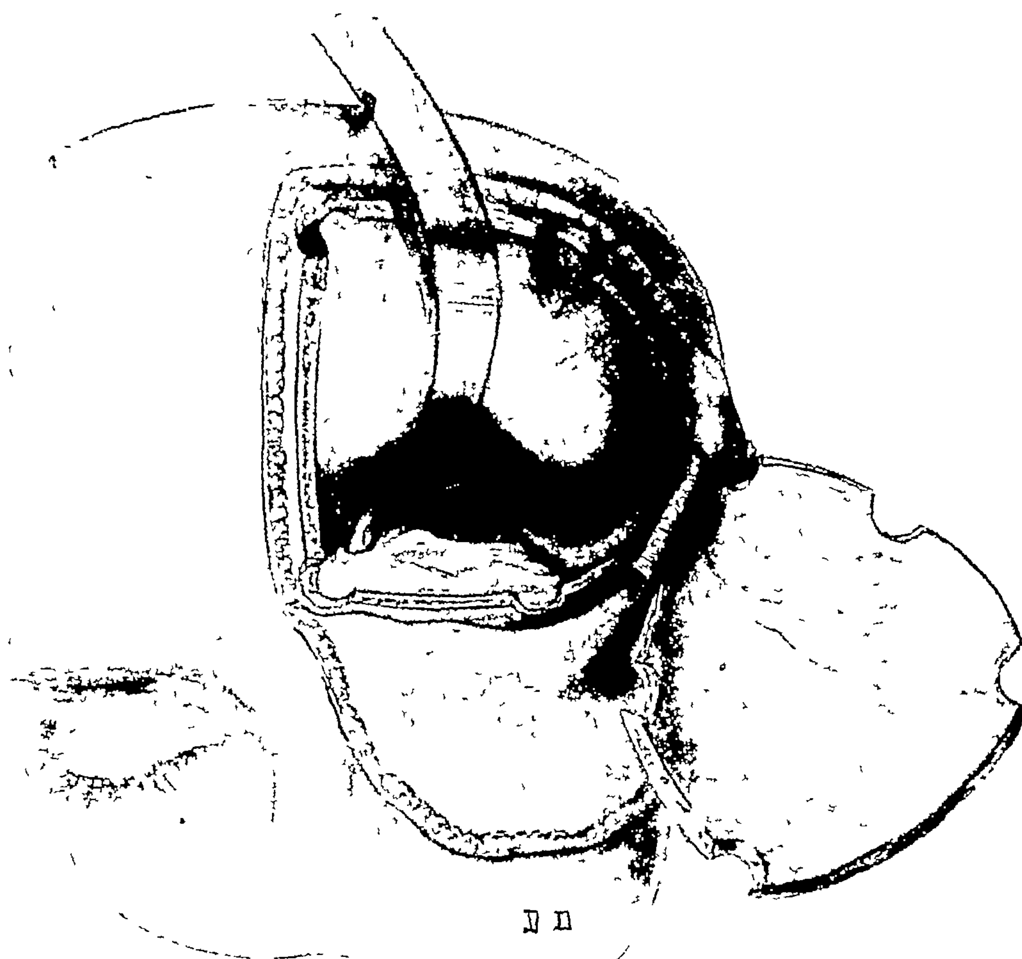


Fig 189

Exposure of the fistula in a case of delayed cerebrospinal rhinorrhœa through a unilateral transfrontal flap.

fracture, since the blood may be coming purely from a ruptured ear drum. The only unequivocal sign that the cerebral membranes have been torn is discharge of cerebrospinal fluid or brain tissue from the external auditory meatus.

Whether a compound fracture of the petrous bone is present or not, but the drum is broken, great care must be taken to avoid infection. To ensure this, coagulated blood should be removed at once from the external auditory meatus by swabbing, but never by syringing. Then a small wisp of carbolic gauze should be placed

in the external auditory canal and a sterile dressing bandaged over the ear Syringing and tight packing of the external auditory meatus are unwise because of the danger of washing infection inwards or of causing blood to collect intracranially When cerebrospinal fluid is present in the discharge, prophylactic chemotherapy must be started to prevent meningitis. Healing of a cerebrospinal fluid fistula may be promoted by limited fluid intake and by daily lumbar punctures Occasionally the tear in the dura mater has to be explored and surgically repaired

Further details of the anatomy and complications of mastoid injuries will be given later

THE DURA MATER

The treatment of open wounds confined to the scalp and skull is a relatively simple matter On the other hand, when the dura and brain have been damaged, strict observance of neurosurgical principles becomes necessary if good results are to be obtained Not only has the immediate problem of life and death to be considered, but possible sequels such as epilepsy and brain abscess must be minimised by correct procedure In cases where there is a rise in intracranial pressure, an injudicious opening of the dura mater, for example, or an imperfectly planned operation on the brain may lead to extensive cerebral damage

When the Dura Mater has not been Torn.—Intact dura should never be opened when the superficial parts of a wound are obviously infected otherwise meningitis or encephalitis will result. It is also dangerous to open the dura when a wound is heavily contaminated Contamination implies the presence of foreign material in a wound, infection, that inflammation due to bacterial action has already started

When a patient is unconscious and a clot can be seen through the dura, it should be evacuated if this can be done aseptically A small incision, not more than ½ in in length is made through the dura, the nozzle of a sucker inserted and the blood removed The dura is then carefully sutured

When a patient is unconscious and intradural tension is increased, as judged by finger tip palpation, and a subdural clot cannot be seen, cerebrospinal fluid should be withdrawn via the lumbar theca until the pressure is reduced to normal When intracranial pressure remains high in spite of lumbar drainage a decompression is necessary, and thus, according to circumstances, may have to be made on the side of the skull opposite to the wound

In a conscious patient, whether a subdural clot can be seen or not, it is not absolutely necessary to open the dura and if there is any doubt about infection it is better not to do so

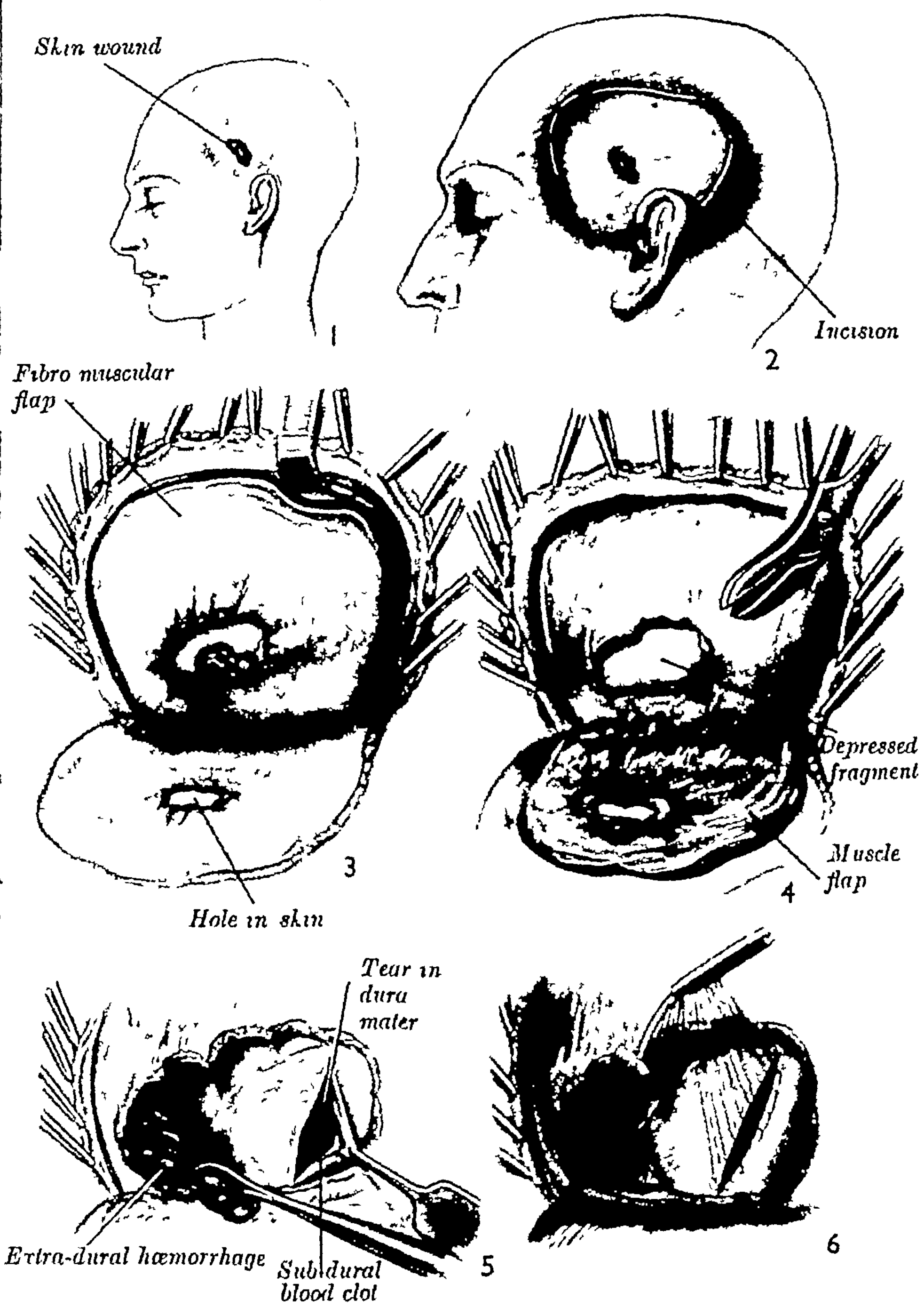


FIG 190

A method of excising a compound dural tear in the temporal region.

1, The site of the wound 2, A semicircular skin flap was used for the exposure
3, The skin flap turned down exposing a tear in the temporal muscle
4, Reflection of the fibromuscular flap subperiosteally
5, Exposure of dural tear and removal of an extradural clot A ruptured meningeal vessel was sealed by diathermy coagulation 6, Repair of dural tear by sutures.

When the Dura Mater has been Torn.—On those occasions when it is known that the dura is torn, a lumbar puncture needle with its stylet in position should be introduced into the lumbar theca before the start of the operation. This necessitates the patient lying on his side, but there is no disadvantage in this

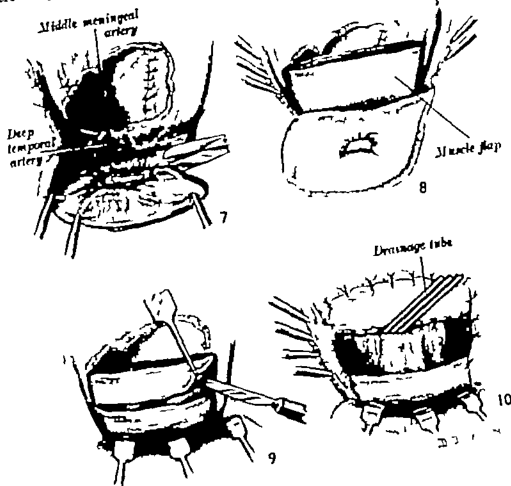


Fig. 101

A method of excising a compound dural tear in the temporal region.

7 Removal of injured part of muscle

8, The shortened temporal muscle left the upper part of the bone defect uncovered.

9 and 10, Reformation of the fibromuscular flap by plastic repair. Finally the skin wound was excised and its skin flap sewn into position with two layers of sutures.

position since his head can easily be manipulated into a position which will make any wound accessible. By withdrawal of the stylet of the puncture needle and spinal drainage, intracranial tension can be lowered at any stage of the operation if found necessary. In this way intradural manipulations may be facilitated and harmful cerebral herniations prevented.

The first stage of the operation is to expose the whole of the

dural tear by removing the bone around it, either by piecemeal nibbling or by block resection When fully exposed the dural tear should be sutured if elastic retraction of the membrane or loss of tissue has not made this impossible (Figs. 190 and 191) A ragged tear may be sparingly excised if diathermy is available to seal off the meningeal spaces afterwards, and such sealing is effected by drawing a diathermy button along the excised dural edge. On no account must dura be extensively excised, as this may reopen meningeal spaces which have already been sealed off by natural processes, or allow a cerebral hernia to form.

When dura has been lost, the defect may be repaired by a fascial or amniotic graft if it is considered that the wound will heal by first intention. However, if there is the slightest danger of infection the skin only should be pulled together and dural repair left for a later operation.

Recently Penfield[1] has shown that amniotic membrane causes less gliosis when in contact with injured brain than pericranium or fascia lata. This, therefore, is the tissue of choice in the repair of dural defects Its preparation is simple. Sheets of amnion are obtained at a healthy birth, sterilised by boiling and stored in alcohol Tendency to crumple may be overcome by preparing it between flat glass plates. At the time of the operation it is cut to the required shape, lain across the defect and teased beneath the dural edges In my experience attempts to suture it in position have not always been successful because it is so friable.

When amniotic membrane is not available and a large graft is required, fascia lata will have to be used. In this case a linear incision is made on the outer side of the thigh and a rectangle or square of fascia removed according to the size of the dural defect. It is trimmed to the exact shape and sewn to the dural edges with its smooth surface facing the brain. Small dural defects may be repaired by pericranium taken from a healthy part of the wound.

THE BRAIN

The special conditions of the brain demand a special operative technique, the details of which must be observed meticulously if a successful operation is to be performed. Manipulations must be carried out with the utmost gentleness and great care taken not to injure healthy brain tissue adjacent to the operative field by heavy retraction as a wound track is opened for inspection. Any kind of rough handling of the brain will lead to swelling, which

[1] Chao, Y C, Humphreys, S S, and Penfield, W "New Method of preventing Adhesions, use of Amnioplastin after Craniotomy" *Brit Med Jour*, 1940, 1, 517

may not only impede surgical procedure but also may prove fatal All bleeding vessels must be sealed either by coagulation, silver clips or by muscle grafts, for until this has been done it is useless to close a wound, since a post-operative clot is certain to collect and compress the brain When a vessel is torn it is unwise to press a finger or a swab heavily on to it to stop bleeding, as consequent displacement of the brain will lead to rupture of distant and inaccessible veins entering the dural sinuses The bleeding vessel should be isolated by suction and sealed by what seems the most suitable method

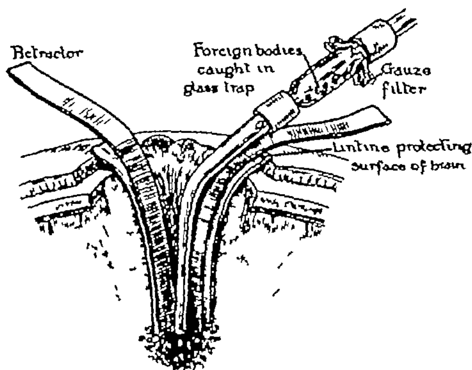


Fig. 191
Débridement of a wound track.

Treatment of a cerebral wound is designed to remove dead tissue, extravasated blood, fragments of bone and foreign bodies, and these objects must be kept clearly in mind and nothing further attempted if unnecessary damage to healthy parts of the brain is to be avoided In superficial wounds, foreign bodies may be picked out with dissecting forceps and loose tissue washed away with jets of warm saline projected from a Canny Ryle syringe. When a deep laceration occurs, the wound track should be opened and exposed in its whole length by means of flat metal brain retractors (Fig 192) To do this successfully, skilled assistance and efficient lighting are necessary Wet lintine swabs placed along the walls of the wound track will protect the brain tissue

from damage by the metal retractors. Débridement is then carried out by suction. A glass tube of 3-mm. lumen diameter is used as the sucker nozzle. This is passed along the opened wound track and weak suction used, so that healthy brain tissue is not lifted at the same time as damaged matter is removed. Dead brain tissue, extravasated blood and small bony fragments will pass along the lumen of the tube, and these should be caught in a suitable trap, otherwise they will block the sucker. Medium-sized foreign bodies will be drawn into the opening of the glass sucker and may be removed when the sucker is withdrawn. Large bony fragments or missiles may be identified and loosened by suction and then removed with forceps under direct vision. Healthy tissue must never be removed in the hope of getting a clean block resection, as this is likely to lead to spread of infection or to unnecessary neural defects. In particular, débridement in the region of the motor cortex must be done conservatively and with great care if hemiplegia is not to be the result.

RETAINED MISSILES

A bullet or bomb fragment is often deeply embedded in the brain, and whether it should or should not be removed immediately depends on its accessibility. When it can be reached safely along a wound track without infliction of further damage to the brain it should be removed, but on no occasion should a separate entrance through the skull be made for its extraction. During débridement a bullet may be encountered in the track of the wound, and in these cases it is extracted with forceps. For extracting from the *bottom of a wound metal fragments which are difficult to grasp* with forceps an electric magnet is useful. The steel bar of the magnet is passed into the wound, the current switched on and after a few seconds the magnet withdrawn A few volts only are needed to activate the magnet, otherwise pieces of metal may be dragged through healthy tissue from a distance, with resulting laceration of the brain tissue. It must be remembered that a magnet is not a powerful instrument to be applied to the surface of the brain but is purely an adjunct to débridement and never replaces it. When a metal missile is not easily accessible, it should be left *in situ*, because it rarely gives rise to a brain abscess and its presence does not materially influence the incidence of epilepsy. An epileptogenic focus tends to lie in the superficial or cortical part of a wound track and not in the area immediately surrounding a foreign body.

Removal of a foreign body at a later date may be necessary if complications such as infection or epilepsy develop. In these cases the operative entrance is determined by accessibility. For

example, a bullet is approached from the surface nearest to which it is lying, provided that this is a silent area. It would of course, obviously be wrong to incise the motor cortex in order to gain access to deep tissues. When infection is already present in the superficial layers of a wound, the only indication for removal of a foreign body is when it is giving rise to an expanding abscess

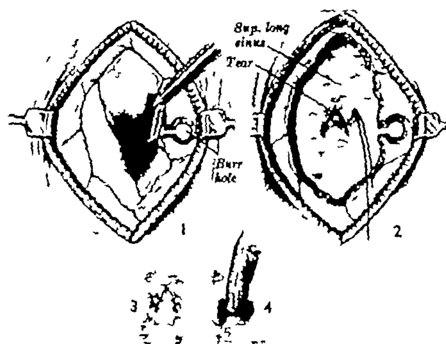


Fig 183

Exposure and repair of a tear of the wall of the superior sagittal sinus.

1 Removal of the bony fragments starting from a burr hole peripherally placed. 2 Suture of the tear in sinus wall. 3, Completion of suturing. 4 The suture lines are finally covered with a muscle graft.

DURAL VENOUS SINUSES

Repair of a torn dural venous sinus is always difficult and often hazardous. Therefore, whenever a depressed fracture is seen to lie over the superior longitudinal or lateral sinus, certain pre-operative precautions should be taken to anticipate bleeding The patient's blood group is determined and a pint of blood obtained pre-operatively After primary shock has been treated, the theatre is fully prepared and the patient, if not already unconscious, is given an injection of morphia. When asleep or quiet, his head and one leg are carefully shaved and made aseptic. The patient is then placed on the table and every means used to lower cerebral venous pressure. His head is raised and fixed above the level of the rest of the body When a neurosurgical table is available the patient should be tilted into a sitting position

The blood at all stages must be fully oxygenated by keeping the tongue forwards and by oxygen bubbled into the nasopharynx through a small tube Local anæsthesia is the method of choice. General anæsthesia by inhalation must always be avoided, as it blows up the venous sinus and often causes serious bleeding before the surgeon is in a position to control it. When a patient is restless intravenous injection of Pentothal is the best method of anæsthesia.

Before commencing the operation on the head the anterior compartment of the leg is opened and muscle grafts prepared. At the same time the internal saphenous vein is exposed and a saline drip started so that, when necessary, blood may be given without delay. Then the superficial layers of the head wound are excised. Bony fragments are lifted out singly, beginning at the point farthest away from the sinus until the dural tear is fully uncovered. One of the great dangers is that severe bleeding may begin before all the bone necessary for exposure has been removed. When this happens it is best to proceed with the bone removal as expeditiously as possible.

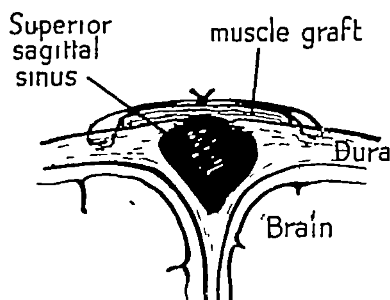


FIG 194
A method of holding a muscle graft in position over a dural sinus by means of suturing

On those occasions when a sinus is found to be completely severed. each end must be clamped with forceps and firmly ligated with stout silk thread. When a tear is extensive but not complete, the edges of the wound should be drawn together with a continuous or interrupted suture and the suture line covered with a muscle graft (Fig. 193). When a tear is small it can often be controlled by muscle grafts alone, but these may have to be sewn into position as shown in Fig 194.

DRAINAGE AND DRESSINGS

A small wound may be closed without drainage after all bleeding points have been sealed and when it is thought to be free from infection. Drainage, on the other hand, is the safer procedure when a wound is extensive or obviously has been heavily contaminated. When it is decided that drainage is necessary, a narrow strip of corrugated rubber or fine tubing is placed beneath the scalp and brought out through what will be the most dependent part of the wound, as judged by the position in which the patient will subsequently be nursed. Intradural drainage must never be employed. Also, drainage tubes should not be allowed to issue from those positions where healing is apt to be delayed. Before

applying sterile dressings, blood should be carefully and completely washed from the scalp to avoid infection The wound is then dressed with layers of gauze soaked in surgical spirit In those cases where the dura has been left widely open the brain must be protected from pressure by building up the dressings at the periphery of the wound over sound bone (Fig 195)

When a tube has been inserted to drain possible post-operative oozings of blood it should be removed within thirty six hours. It should, however be left in as long as necessary when infection

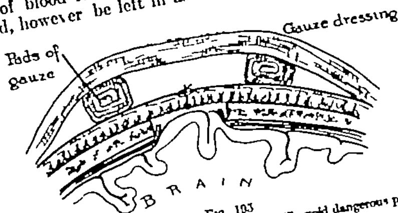


Fig. 193
When bone has been lost a built up dressing will avoid dangerous pressure on the brain.

is expected In the absence of infection the opening left by a drainage tube ought to be closed with a suture to promote rapid healing and to avoid the formation of a fistula. When a wound suppurates, free drainage must be provided at once, if necessary through suitable stab incisions, since pus under tension will lead to spreading encephalitis. When the dura mater is open and cerebrospinal fluid or a brain hernia is stretching a sutured wound, repeated lumbar punctures will relieve the tension and allow the skin to heal firmly

CEREBRAL FUNGUS

A cerebral fungus is a protrusion of brain substance through a defect in all its coverings, through the meninges, the skull and scalp It is a most troublesome complication and difficult to cure Often it results from faulty or inadequate surgery in the first instance, and in particular appears in the type of wound in which there has been loss of all the covering tissues of the brain and where the scalp has been pulled together under tension Infection of the wound leads to suppuration, the skin wound fails to heal or breaks down secondarily, and the brain begins to bulge Why the brain should progressively bulge through

a dural defect is matter for argument, since under normal conditions the ventricular or intracranial pressure is about equal to the atmospheric.

The following are the main theories of the etiology of cerebral fungus :—

1. Infective—local encephalitis, abscess or foreign body leads to swelling, the mass in each case being increased by associated cerebral œdema.
2. Increase in brain volume, due to distension and pulsation of the cerebral arteries.
3. Hydrocephalus resulting from obstruction of the circulation of the cerebrospinal fluid by basal adhesions. In such a case there is a progressive distension of the ventricular system



FIG 196
A large cerebral fungus due to faulty surgery

4. Strangulation of the cerebral veins at the periphery of the fungus at the dural or bony edge. This leads to cerebral congestion, to œdema and swelling in the tissues of the fungus.[1]

O'Connell [2] criticises these theories and offers an alternative of his own. First, he points out that the ventricle on the affected side dilates as a result, presumably, of tissue atrophy consequent on the trauma. Then, with the rhythmic rise in the intraventricular tension, as a result of arterial pulsation and respiration, the ventricle dilates progressively and bulges into the hernia, causing its increase in size.

Treatment—*Prevention.*—Prevention of cerebral fungus is much easier than cure. Progressive herniation of the brain never occurs if deep infection is avoided and a sound repair of the

[1] Leriche, R *Lyon chir*, May-June 1916
[2] O'Connell, J E A. "Traumatic Cerebral Fungus" *Brit Jour Surg*, 1943, 30, 201

scalp is obtained In scalp defects early epithelialisation of a wound will overcome any tendency to cerebral bulging

Cure —Given that a cerebral fungus is present, its cause must first be determined, in particular, the possibility of a deeply seated cerebral abscess must be eliminated This can be done satisfactorily and easily by means of encephalography or ventriculography When an abscess is discovered it must be drained or excised, otherwise the hernia will never recede nor the wound heal satisfactorily

If an abscess is not present, every effort must be made to get the wound covered by epithelium Persistent surface infection which prevents epithelialisation, such as chronic osteomyelitis or infected foreign matter embedded in surface fibrous tissue, must be correctly treated as soon as possible

Raised intracranial pressure is controlled by repeated lumbar puncture and drainage Deep infections are treated by oral sulphonamide or systemic penicillin therapy Plastic repair of the scalp defect is rarely feasible or wise On the other hand, Thiersch grafting is safe and often valuable On no occasion must amputation of a cerebral fungus be attempted, as spreading encephalitis or meningitis will almost certainly be the result Even if removal were apparently successful, the result will only be temporary, since herniation of further brain tissue will occur if the underlying cause still remains

MENINGITIS AND ENCEPHALITIS[1]

Brain tissue possesses considerable bactericidal powers and is able to overcome mild infections or to localise suppurative processes by the formation of barriers of neuroglial and fibrous tissue Thus penetrating wounds of the brain do not necessarily lead to a meningitis or to a spreading encephalitis In particular, the spread of infection over the cortex is soon limited by the obliteration of the subarachnoid spaces either by swelling of the brain or by meningeal adhesions in the region of a wound Unfortunately, the natural barriers against infection cease at the ventricular walls, and once organisms gain access to the ventricular cavities they are freely washed into the cerebrospinal fluid spaces and diffuse meningitis results (Fig 197)

When an infective intracranial complication develops, lumbar puncture should be done at once and a specimen of cerebrospinal fluid sent for bacteriological and cytological examination Suitable and extensive chemotherapy is started as soon as a diagnosis of meningitis or cerebritis is made and this is supplemented later by suitable serum injections when the causative organism has been

[1] Ureck, E. "Valeur de la Seroprophylaxie des Meningites Post traumatiques." Schweiz med Wschr 1943, 73, 115

isolated. Also, cerebrospinal fluid should be freely withdrawn twice daily by lumbar punctures. After the second puncture this is usually a tedious procedure, since fluid is apt to drain away very slowly owing to increase of its viscosity consequent on the presence of products of inflammation. Often it takes half an hour to collect more than 30 c.c , but it is time well spent since drainage relieves pain and encourages the circulation of the cerebrospinal fluid. Cisternal puncture may be used as an alternative route for drainage when the lumbar theca becomes blocked with adhesions,

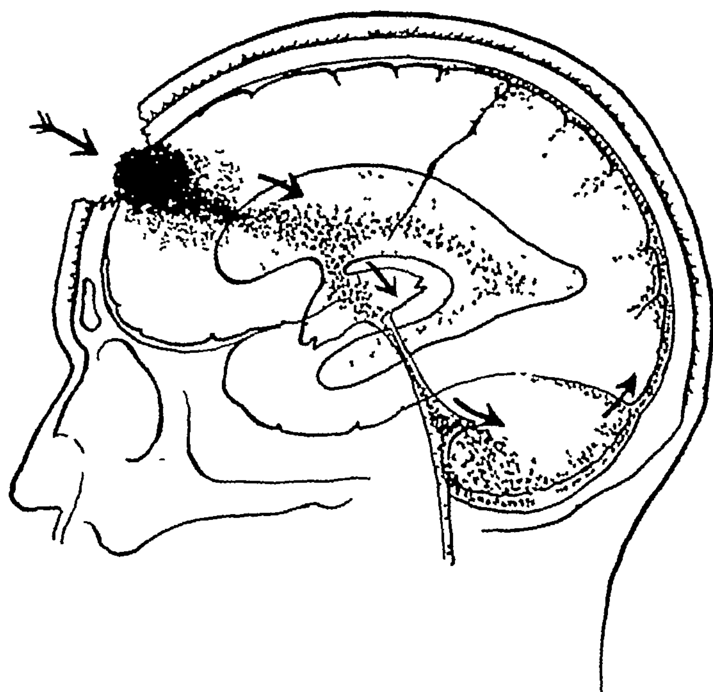


Fig 197

Ventriculitis is the common cause of a fatal termination resulting from an infected wound of the brain (*After Jefferson*)

as it so often does. Toxæmia is treated by the administration of copious amounts of fluids; when a patient is vomiting and unable to drink freely, a cannula should be tied into a vein and glucose saline given as a continuous drip.

It is important not to give excessive amounts of fluids intravenously, otherwise transudations will occur into the pleural or peritoneal cavities with fatal results. For this reason not more than 5 pints of fluid should be given in one day. Improvement or retrogression may be judged in cases of post-traumatic meningitis not only on clinical signs but also on the information given by cell counts done at the time of each lumbar puncture. Even in fulminating cases of infection many lives can be saved by persistent and energetic treatment, and no case should be given up as lost until every known type of therapy has been tried.

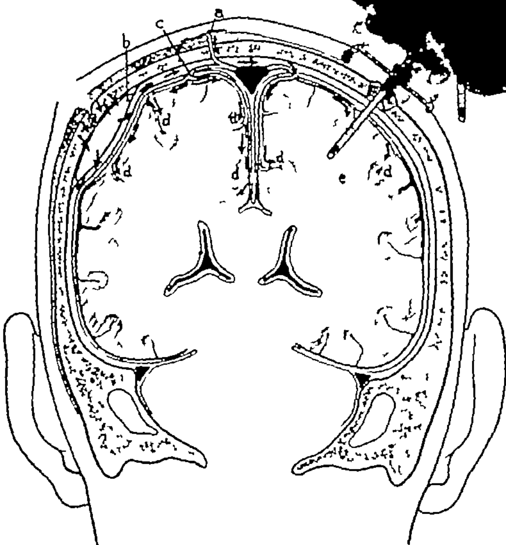


Fig. 109

Several modes of spread of infection in wounds of head

A scalp wound has become infected. (1) The subaponeurotic areolar layer may become seat of cellulitis which may reach emissary vein (a) and cause infective thrombo-phlebitis of it which may spread intracranially. (2) Infection may spread through torn pericranium, Haversian canals of bone or fracture if present to extradural plane—extradural abscess (b). Infection may spread in small veins of dura and involve a cerebral vein in its passage through the dura. Infective thrombo-phlebitis of a cerebral vein (c) may cause meningitis as it spreads inwards, and/or cerebral abscess as tributary veins (d) draining brain substance become affected. The infection may spread through dura of the walls of a dural sinus (with or without thrombosis of sinus) and so reach the opposite side where similar spread along cerebral veins may take place. Method of drainage of a recently formed cerebral abscess (e) by rubber catheter supported by a disc at scalp surface is also shown. (*After Dott.*)

CHEMOTHERAPY

cs in chemotherapy both during and since the complete rewriting of this section articular importance are (*a*) the sulphonamides, d (*c*) streptomycin

object of chemotherapy is to prevent the development of on which a wound has become contaminated and to limit and cure infection when once it has developed.

In every case of obvious compound injury of the brain, and also when internal compounding is presumed to have occurred, prophylactic measures to prevent infection should be instituted. It is, of course, impossible and, in fact, unnecessary to isolate every organism in every wound that might have been contaminated; on the other hand, it is essential for the correct treatment of an infection to discover the nature of the causative bacterium and to know to what drugs it is sensitive Lack of such knowledge may well lead to medico-legal difficulties in the future

PROPHYLAXIS

1. **Internal Compounding.**—When a paranasal fracture is not severe, as shown by minimal bruising about the eyes and by the small amount of bleeding from the nose, a full course of sulphamethazine is given. When the patient cannot or will not swallow, a course of penicillin is given instead.

When a paranasal fracture is severe, and particularly in cases of cerebrospinal fluid leakage, a full course of sulphadiazine by mouth and penicillin by muscular injection are prescribed immediately. Therapy must not be delayed on any pretext, since a fulminating meningo-encephalitis may develop within an hour.

2. **Compound Fractures Necessitating Open Operation.**—After cleansing of the skin and débridement of the depths of a wound a decision has to be made whether the raw surfaces themselves should be treated by chemical methods before the wound is closed My own procedure is as follows When the wound is largely cranial and the brain not seriously damaged, and when contamination is not heavy, the raw surfaces are sprayed by means of a syringe and a fine-bored needle with a solution of sodium penicillin, 50,000 units being used in 5 c c water. The wound is then closed and the patient given a full course of systemic penicillin and sulphadiazine.

When the brain has been severely damaged and contamination is heavy, the superficial part of the wound after débridement is washed out freely with a warm solution of 10 per cent. Milton.

This procedure not only stops troublesome oozing from the small vessels but possibly also destroys gram negative organisms that may be present and give rise to infection later in spite of sulphonamide and penicillin therapy In the near future streptomycin or some such preparation may well supersede Milton After being syringed with Milton solution the wound is washed freely with sterile normal saline solution The wound is gently mopped and the raw surface dusted finely with penicillin and sulphonamide powder Only in those cases of complicated paranasal fractures where a cavity between the dura and the top of the nose has been left should a fine drainage tube be left in position so that penicillin may be injected for two or three days In fractures of the vault, tubes are best dispensed with and the wound firmly closed so as to minimise the danger of infection by gram negative organisms A full course of sulphonamides by mouth and penicillin by intramuscular injections are also given in this type of case

WHEN INFECTION HAS DEVELOPED

As stated above, it is essential to determine the nature of the causative organism if correct treatment is to be instituted It is, of course, just as wrong to delay treatment until the bacteriological facts have been established In the case of a head injury when infection has occurred all that is necessary is a lumbar puncture and bacteriological examination of the cerebro spinal fluid Chemotherapy can then be justifiably started before the bacteriological examination is completed

For correct procedure certain principles and facts must be constantly kept in mind —

(a) Chemotherapy does not counteract toxaemia replace lost fluids or prevent inanition, nor does it assuage pain, allay anxiety or promote sleep All the customary rules of surgery must, therefore, also be observed in combating the effects of infections general measures are, indeed, all the more essential since the action of the drugs themselves adds a further burden to those of the toxaemia of infection

(b) Penicillin and sulphonamides do not directly kill bacteria, they merely prevent their multiplication and it is accordingly essential to maintain the drugs *continuously* at an adequate concentration in the affected tissues Inadequate concentration may lead to the development of resistant organisms.

(c) Antibiotics are active only if they can come into physical contact with the invading bacteria, they cannot pass

adhesive blocks in the arachnoidal pathways nor pierce the walls of abscesses.

(*d*) Penicillin does not easily cross the blood-brain barrier; to obtain and maintain an adequate bacteriostatic level in the cerebrospinal fluid the preparation must therefore be instilled in the cerebrospinal fluid spaces either by lumbar, cisternal or ventricular puncture

(*e*) In meningo-encephalitis the infecting organism may settle outside the intracranial cavity and the primary source of infection be susceptible to systemic chemotherapy; alternatively, an infective lesion primarily in the brain may give rise to a septicæmia. Therefore in all cases of pyogenic meningo-encephalitis both systemic and intrathecal chemotherapy are necessary.

PENICILLIN

Systemic.—In the systemic administration of penicillin the only danger is that of underdosage. The dangers of insufficient dosage are: (1) the drug may be excreted so quickly that the bacteria are allowed to multiply unhindered for long periods, and (2) resistive strains of organisms may develop The only criticism of large doses of systemic penicillin is that they are wasteful. Once the level of 0 1 units per cubic centimetre in the blood has been reached no more good can be obtained by increasing the concentration. In prophylaxis, the danger of giving insufficient dosages must be avoided; adequate dosage is the essence of successful penicillin therapy.

Doses

Curative.—15,000 units every three hours
Prophylactic.—200,000 units night and morning

Intrathecal.—Penicillin may be injected intrathecally into the lumbar or cisternal spaces or into the lateral ventricles.

Doses

Curative.—In intrathecal therapy there is a danger of injecting too strong a solution. This would lead to damage to the cells lining the arachnoidal membranes and the aqueduct of Sylvius, with resulting adhesions, strictures and hydrocephalus. 12,000 c c. twice daily are sufficient to keep up an adequate continuous level in the cerebrospinal fluid.[1]

[1] Cairns, H "Penicillin in Suppurative Lesions of the Brain and Meninges" *Brain*, 1947, **70**, 251

Prophylaxis.—For prophylactic purposes in head injuries the action of the sulphonamides is depended on; it is neither necessary nor wise to subject a patient to the dangers of intrathecal manipulations.

Routes of Administration[1]

Lumbar.—When the arachnoid pathways are freely open penicillin injected by the lumbar route will readily diffuse over the whole of the cerebrospinal space, including the ventricular cavities. The lumbar route for administration of the drug may, therefore, be used so long as the flow of cerebrospinal fluid from the puncture needle remains free.

Cisternal.—Injections into the cisterna magna are necessary when the spinal thecal canal becomes blocked by arachnoidal adhesions.

Ventricular.—By means of a suitably placed burr hole a fine rubber tube can be passed into the anterior horn of a lateral ventricle. Such a tube may safely be left in position for seventy two hours. After this period the danger of cross infection becomes so great that it has been my practice to remove the tube, to close the wound and to tap the opposite ventricle whenever it is necessary to continue the intraventricular therapy. The indications for ventricular therapy are :—

(*a*) When the other routes have become blocked by adhesions.
(*b*) When for other technical reasons it is impossible to use the lumbar or cisternal route.
(*c*) When in open head injuries the ventricle has been perforated and contamination has been severe.
(*d*) When it is thought that an intracerebral abscess has burst into a ventricle.
(*e*) When, in spite of lumbar and cisternal administrations, the patient's condition does not improve even though the infective organism is sensitive to penicillin.

STREPTOMYCIN[2]

The antibiotic streptomycin is now being used both systemically and intrathecally in the treatment of infections due to organisms that are relatively or absolutely resistant to penicillin and to sulphonamides. So far the optimal dosage and best routes of administration have not yet been fully determined. These difficulties will no doubt soon be overcome and probably more efficient antibiotics will be discovered.

[1] Logue, V., and McKissock, W. "Further Reduction in Hospital Infection of Wounds." *Brit. Med. Jour.*, 29th September 1945, p. 413.
[2] Smith, H. V., Vollum, R. L., and Cairns, H. "Treatment of Tuberculous Meningitis with Streptomycin." *Lancet*, 24th April 1948, p. 627.

SULPHONAMIDES

Of all the sulphonamides, sulphadiazine is the one that most easily passes the blood barrier to produce an antibacteriostatic level in the cerebrospinal fluid. Its main disadvantage is that it may be precipitated in the glomerular apparatus of the kidney and destroy its function.

To avoid this complication copious fluid should be administered together with doses of alkali such as sodium bicarbonate. To be safe a patient must pass at least 1,500 c.c. urine per day. Other dangers are those common to sulphonamide therapy in general. In prophylaxis, when infection has not yet reached the intracranial cavity, sulphamethazine instead of sulphadiazine is used.

DOSAGE OF SULPHONAMIDES[1]

SEVERE INFECTIONS (ENDANGERING LIFE)

	Adults	Children		
		1 to 3 Years	4 to 10 Years	11 to 15 Years
Initial dose	2 to 4 grm by mouth	0 5 grm by mouth	1 grm by mouth	1 to 2 grm by mouth
Followed by first period —2 to 3 days	1 5 grm 4-hourly	0 5 grm 4-hourly	0 75 grm 4-hourly	1 grm 4-hourly
Second period—2 days (approximately two-thirds of dose of first period)	1 grm 4-hourly	0 5 grm 6-hourly	0 75 grm 6-hourly	1 grm 6-hourly
Third period—2 days (approximately one-third of dose of first period)	1 grm 6-hourly	0 25 grm 6-hourly	0 5 grm 6-hourly	0 5 grm 6-hourly

GRAM-NEGATIVE INFECTIONS[2,4]

Since the advent of chemotherapy and the control of diffuse infections of the cerebrospinal fluid spaces caused by penicillin-sensitive organisms, the study of the bacteriology of penetrating

[1] Stammers, F A R "Chemotherapy" *Brit Surg Practice*, 1948, 3, 44
[2] Lewin, W "Gram-negative Meningitis following Head Wounds" *Brit Jour Surg*, January 1948, 35
[3] Wilson, C "Streptomycin in Non-tuberculous Infections" *Lancet*, 18th September 1948, p 445
[4] Lewin, W, and Vollum, R L "Streptomycin Treatment of Meningitis due to Gram-negative Saprophytes" *Lancet*, 18th September 1948, p 446

wounds has made it clear that meningitis and troublesome local infections occasionally result from gram negative organisms. The three organisms chiefly concerned are (a) coliform bacilli (lactose-fermenting gram negative bacilli), (b) *Pseudonema pyocyaneus* and (c) *B. proteus*.

Treatment.—In infections due to gram negative bacteria the local source of infection must be removed surgically if it can be discovered. There is no specific drug that will combat the infection, but sulphadiazine so far appears to be the most valuable. The value of streptomycin is at present under investigation.

SUMMARY OF THE LESSONS LEARNED DURING THE WAR[14]

The lessons learned from the 1939-45 war were partly new and partly confirmations of principles gained from earlier experiences. One decision of fundamental importance was to segregate certain forms of injury. This necessitated the training of surgeons for the work and fortunately brought into the neurosurgical field many who were already knowledgeable in other branches of surgery, a benefit not only for themselves and for neurological surgery, but also for men whose main activities in peace time had been those of neurological surgery.

The services of men engaged in the academic sciences were recruited in the solution of technological problems, a liaison which soon led to the understanding of each others' difficulties, strengths and weaknesses. An attempt was also made to foresee the nature of future warfare and to anticipate its difficulties. This led to the formation of mobile teams and made possible early and complete or definitive surgery on a large scale. Finally, knowledge gained from early experiences was readily pooled and discussions of problems took place at all levels. With these advantages, neurological surgeons set forth to meet problems and made many technical advances.

The Scalp.—In the first instance it was realised that by far the best barrier against infection is epithelium: every effort was therefore, to be made to close a wound completely and to promote primary healing. In case of skin loss, plastic procedures were freely used to cover defects in the bones and with tissues of such thickness that they could safely be lifted on a future

Brit. Jour. of Surg. War Surgery Supplement No. 1. "Wounds of the Head."
Lumsden, R. B. "War Wounds and Injuries involving the Paranasal Air Sinuses." *Edin. Med. Jour.*, 1945, 52, 402.
Brit. Med. Bull., 1945, 3, Nos. 1-2.
Wertheimer, L. G. "Observacoes Anatomo-Clinicas de Traumatizados Cranio-Encefalicos." *Revista do Hospital das Clinicas*, 1946, 1, 71.
Wertheimer, L. G. "Traumatismos Cranianos." *Arquivos de Cirurgia Clinica e Experimental*, 1945, 9.

occasion should this be necessary to repair a bony defect or to excise an abscess or meningo-cerebral scar. Furthermore, the shapes of wounds were fashioned not only to give adequate exposure of deeper tissues, but also with an eye on future possible reopening. In the main, the standard of surgery of the scalp in the past war was excellent and to a large extent accounted for the good results that were obtained.

The Skull.—In the treatment of paranasal air sinus injuries there were two schools of thought, the radical and the liberal :—

(*a*) In compound wounds the radical school advised complete exenteration of the damaged sinus including the mucous membrane When in such cases the dura was opened damaged brain tissue was also freely sucked away before the dura was repaired. Similar operations were readily embarked upon in the acute stages in cases of internal compounding when the dura was thought to be torn or when X-rays revealed a displaced fracture of the ethmoid plate. Indeed, so keen did the school become on open repair that many new and ingenious X-ray shoots were designed to detect possible fracture.

(*b*) In the liberal school, the main object in open wounds affecting the paranasal air sinuses was to repair the dura and largely to leave the splintered air sinuses to the care of natural processes and, in my opinion, that was the wiser choice. In cases of internal compounding operative treatment was reserved for those cases where a cerebrospinal fluid fistula persisted for many days and the leakage of cerebrospinal fluid was profuse. This again I believe is a wise choice. Delayed cerebrospinal fluid fistula, on the other hand, necessitates immediate surgical repair.

The Brain.—One of the more technical advances of the last war was the thoroughness with which foreign bodies, chips of bone, blood and pulped brain tissue were removed from the track of a penetrating wound. Not only did these measures save many lives but are one of the main reasons why delayed sequels, such as abscesses and epilepsy, have been so infrequent up to the time of writing.

Resuscitation.—Resuscitation not only saved lives but minimised morbidity. Prolonged shock and low blood pressure not only causes irreversible damage to the body but so lessens resistance that infection more easily supervenes. The early transfusion of whole blood or of plasma is now an established method of treatment

Infection and Chemotherapy.—With the advent of chemo-

therapy the success of primary and firm closure of a complex head wound was enhanced The war provided full opportunities for determining the optimum dosages and the best routes of administration both for the prevention and for the combating of infection. It soon became clear that to be efficacious a drug must come into direct contact with the infecting organisms As the infections due to certain well known germs were satis factorily controlled by chemotherapy the action of lesser known bacteria was brought to light, and in particular, the behaviour and importance of a gram negative group was discovered and recorded It has been made abundantly clear that in infection careful bacterial examinations must be made if correct methods of treatment are to be instituted, in fact, omission of this investigation may soon be regarded as malpractice. In the treatment of infection, chemotherapy alone is, however often insufficient and the source of infection must, when possible be removed by surgical means. Finally, it has been made clear how important the subdural space is in the spread of infection, how difficult the space is to drain and how difficult it is to maintain an adequate penicillin level in it.

Rehabilitation and Psychiatry—The value of medical rehabili tation of head injuries has been established beyond doubt. Good facilities for rehabilitation more than pay for their cost in curtail ing morbidity and in getting men back earlier to their work The contribution that psychiatry has made to the understanding and handling of the post-concussional syndrome has also become obvious. The value of early and correct medical rehabilitation is one of the greatest lessons learned from the past war

The Problem of the Closed Head Injury—That the brain was injured by its movement in relation to the skull and by one part of the brain in relation to another was known before the last war It was well known that the forces of throwing and stopping without impact of the head can cause concussion. Such forces, of course are better named those of acceleration and deceleration It was also known that the brain could spin within the skull and, therefore be subject to damage Such damage is more accurately described as resulting from the forces of rotational acceleration and deceleration To physicists and physiologists we are greatly indebted for their recent contributions to the physics of cerebral injury, indeed I now believe that the physics of the skull/brain system has become an exercise in pure mathematics rather than of actual experimentation There still remains however, one essential gap in our knowledge and that is the physics of a single neurone Again I wish to stress that the physics of injury are not synonymous with the pathology of concussion

The problems that still remain to be solved are as follows :—

(*a*) What is the locus of the damaged neurones that account for the symptoms and signs met with in the acute, subacute and chronic states of cerebral injury ?

(*b*) What is the exact nature of a neuronal injury of submicroscopical dimensions ?

(*c*) At what point does an injury to a neurone become irreversible and what factors are likely to enhance or speed recovery ?

(*d*) What is the relative importance of the direct injury to a neurone compared, say, with the indirect injury brought about by ischæmia ?

(*e*) How can permanent neuronal dysfunction be minimised ?

(*f*) What is the underlying pathology of the so-called post-concussional syndrome ?

A symposium of the main neurosurgical work done by H.M. Forces during the recent war has been published by the *British Journal of Surgery* and contains a comprehensive bibliography.

SUMMARY OF PRECEDING CHAPTER

It may be of value at this juncture to reconsider the principles discussed in this chapter and to attempt to present them in their true perspective

Firstly, it must be realised that in any wound of the head the essential problem is to discover what damage has been done to the brain and mind Even in trivial injuries great psychological trauma may occur, as patients invariably look upon an injury to the head as something sinister The results of physical injury only, however, have been discussed here, as those of psychological trauma will follow in a later chapter

In a case of an open wound allied with concussion, the surgeon is more particularly concerned with the latter state owing to the difficulty in diagnosing its underlying pathological cause The nature and extent of the wound itself is a much easier problem therapeutically, since this can definitely be seen as exploration proceeds

In considering the wound itself it can be reviewed in terms of (1) the scalp, (2) the skull and (3) the brain

The scalp heals well and, in the absence of loss of tissue, complete primary healing should be obtained in about 100 per cent of cases The skull, on the other hand, does not combat infection efficiently, and although acute osteomyelitis is rare, chronic osteomyelitis is far too common In débridement, therefore, the skull should be liberally excised This is permissible, since repair of the skull may be effected easily and satisfactorily at a later date The only occasion on which infection is likely to occur after careful débridement is when the paranasal sinuses have been opened by fracture, since complete removal of mucous membrane is difficult in such cases

Operations on the dura and brain should always be on the conservative side owing to the danger of removing healthy and undamaged tissue unnecessarily Careful aseptic precautions should be taken when wounds are dressed, and sutures should be removed early to prevent stitch infection To promote healing in a

wound under tension owing to raised intracranial pressure repeated lumbar punctures should be performed. Also chemotherapy should be used to prevent infection.

Experience during the past war taught us that the best results in the treatment of open wounds are obtained if the following principles are observed:—

(i) Early and efficient resuscitation before a major operation is carried out.

(ii) Complete shaving and careful cleaning of the scalp.

(iii) Early definitive operation, which means that adequate neuro-surgical facilities are at hand.

(iv) When débridement has not been complete the scalp should not be sutured.

(v) Removal of all indriven bony fragments and pulped brain tissue.

(vi) Correct usage of sulphonamides and penicillin.

(vii) Immediate closure of the dura mater if necessary by fascial grafts, etc.

(viii) Complete closure of the skin without tension; when there has been loss of tissue it is often necessary to swing two large skin flaps to cover the defect.

(ix) Avoidance of cross infection by correct surgical procedure.

(x) Tubes should not be used in cerebral wounds, either for drainage or for instillation of penicillin, as these open a route for the introduction of penicillin-resistive bacteria.

CHAPTER VII

TRAUMATIC OSTEOMYELITIS

THE bones of the vault of the skull are composed of two tables of compact osseous tissue enclosing the diploe (Fig. 199). Diploe is merely another name for cancellous bone. The only way in which this differs from the medulla of other bones is the presence of large venous channels which course through it. These, as far as I have been able to discover, have no lining

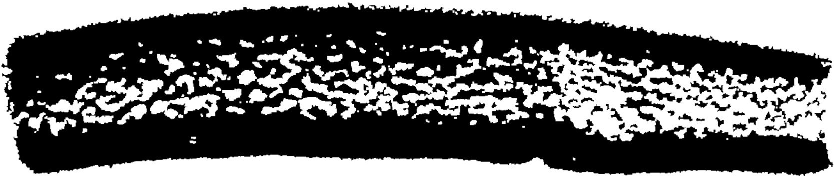


Fig 199

It is along the cancellous tissues of the diploe that infection travels in the bones of the vault of the skull Moreover, it is this sponge-like meshwork that makes surgical drainage so difficult

membrane of soft tissue. Also, they are concerned rather more with the circulation of the brain and scalp than with the metabolism of the bone itself. They drain (i) into the dural venous sinuses, (ii) into the meningeal veins and (iii) into the veins of the scalp. It will be seen, therefore, that there are wide channels of access for possible infection directly from the scalp to the brain (Fig. 200). This is the reason why even simple wounds of the scalp should be treated carefully.

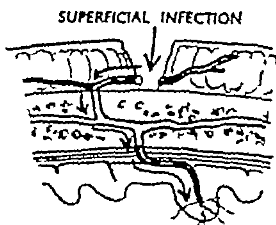


Fig 200

Infection can travel long distances along the diploic channels In particular simple infections of the scalp can give rise to serious intracranial complications

The outer surface of the bone is covered by pericranium. This membrane separates easily, except at the suture lines where it is firmly attached. The endosteum or inner lining consists of the outer layer of the dura mater. This also strips readily, except at the margins of the basal foramina and at the points of entrance of nutrient vessels.

Osteomyelitis of the vault[1,2] is the subject with which we are concerned here, but it is a much less serious complication than the infective complications which follow injuries of the para nasal air sinuses and petromastoid bones which have been dis cussed in Chapters V and VI

Acute fulminating osteomyelitis associated with toxæmia, septicæmia or pyæmia, such as arises in the metaphysis of long bones in young boys, is almost unknown in the vault of the skull Occasionally a severe osteomyelitis of the frontal bone may result from an acute suppurative frontal sinusitis, but this rarely follows injury

OSTEOMYELITIS FOLLOWING CLOSED INJURIES

Osteomyelitis of the vault of the skull following injuries in which the scalp remains intact is rare When it does occur it is always of the chronic or subacute variety The sequence of events is as follows a hæmorrhage occurs between the outer table and its covering membrane, with the resulting formation of a localised subpericranial clot. This becomes infected either from a damaged hair follicle or through the blood stream An abscess occurs, with the result that the superficial layers of the outer table become infected Clinically this process declares itself as a boggy swelling beneath the scalp to form a typical Pott's puffy tumour If left undisturbed the abscess enlarges, bursts through the skin and a fistula results

Treatment in these cases consists in opening the wound widely, draining the abscess and removing any loose flakes of bone The wound is then packed with powdered sulphanilamide or sulpha pyridine or penicillin powder and allowed to granulate from its base Measures to improve the general health should also be instituted since a Pott's puffy tumour is evidence of poor tissue resistance to low grade infection.

If healing does not rapidly take place the following conditions should be suspected (i) tuberculous osteitis, (ii) syphilitic osteitis, (iii) metastatic tumours and (iv) hæmapoietic diseases.

Aseptic Necrosis.—As the name implies, aseptic necrosis is not an infective process It occurs in children and is due to the destruction of an epiphysis by trauma, leading to large defects in the skull

OSTEOMYELITIS FOLLOWING OPEN INJURIES

Direct contamination of an open wound is the usual cause of traumatic osteomyelitis Its occurrence depends almost entirely

[1] Wilensky A O "Osteomyelitis of the Skull." *Arch Surg.*, 1933, 27 83.
[2] Adelstein, L. J., and Courville C B "Traumatic Osteomyelitis of the Cranial Vault with Particular Reference to Pathogenesis and Treatment." *Arch Surg.*, 1933, 26, 539

on the efficiency with which compound fractures of the skull are treated in the early stages. With adequate facilities and early treatment, primary healing in air-raid casualties and civil injuries is somewhere about 100 per cent

Localised Osteomyelitis.—There are three clinical types in this group .—

1. A wound that has apparently healed becomes sensitive to pressure. Then a swelling appears, a length of the scar opens and a discharge of pus occurs. There are no signs of toxæmia, but the wound in spite of careful dressings continues to discharge.

Radiography, though often negative, may show the presence of a foreign body or of a superficial erosion of the outer table.

In such cases the wound should be widely opened if it does not heal rapidly. Commonly, foreign bodies, such as dirt, grit, hair or pieces of clothing, will be found in the deeper tissues These are removed and granulation tissue is cut away. If the surface of the bone is roughened it should be scraped and small sequestra removed. The wound is then packed with sulphonamide powder as described above, or treated by the Bunyan technique. In the latter case the wound at the end of débridement is syringed with one pint of Milton solution, starting with a strength of 1 in 20 and continuing with 1 in 40. The wound is then covered with a sheet of sterile resin-impregnated silk strapped in position on to the surrounding healthy skin. This method in my cases has given excellent results (Figs. 201 and 202 (*a*) and (*b*)).



(*a*) (*b*)

FIG 201

In Bunyan's technique an open wound is covered by a sheet of oiled silk strapped in position as shown in the photograph This method eliminates the necessity of repeated gauze dressings and minimises injury to epithelialisation

2. Sometimes as a result of infection a wound is not consolidated by primary healing. When infection is thought to arise in the deeper tissues, as judged by a copious amount of discharge and surrounding œdema, then the wound should be widely opened and treated as in (1) above. To wait for any length of time in the hope that the wound will heal without further intervention is usually a waste of time. Simple infection of the edges of a skin wound shows signs of improvement within a few days

3. Occasionally the edges of a defect in the skull become infected, resulting in a sclerosing osteitis and a persistent discharge

of pus through one or more fistulæ In these cases radiography shows that the edges of the bone are irregularly sclerosed At times discrete sequestra are demonstrated

Treatment consists either in block resection of the infected bone or removal by piecemeal nibbling In most of the cases it will be found that conservative measures only are necessary to clear up the discharge Whichever method is used when operating great care must be taken not to pierce the dura, as meningitis may be the result. To avoid opening the meningeal spaces the dura should be separated carefully from the bone by means of a curved dissector The amount of bone to be nibbled away can be judged by the extent of the dural granulation tissue,



(a)



(b)

Fig 202

A case of superficial localised osteomyelitis (a) before treatment and (b) after sequestrectomy and the Bunyan treatment.

which must be completely uncovered in all directions Unhealthy granulation tissue is removed by gentle sweeps with a gauze swab

Infection of Loose Fragments.—In the débridement of large compound wounds it is sometimes justifiable to leave loose fragments of bone in position so as to minimise the resulting defect. If at any time when this has been done, infection occurs the wound should be reopened immediately and the loose fragments removed After packing of the wound with sulphonamide large skin flaps may be loosely resutured if an adequate opening say 1 in or so, is left for drainage Small fragments of bone are often inadvertently left behind in unskilled débridement and these commonly give rise to infective complications long after the scalp wound itself has healed Such fragments, of course, should be removed surgically (Fig 203).

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(a) Fig [illegible] (b)

A case of superficial localised osteomyelitis (a) before treatment and (b) after sequestrectomy and the Bunyan treatment.

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Dural and Intradural Abscesses.—In some cases in which a wound has been opened a dimple or thickening of the dura will indicate the presence of an intradural abscess which was not suspected on clinical grounds By gentle finger pressure on the surface of the brain a bead of pus may be made to well up through a fine fistula. When this happens the opening should be enlarged and the underlying abscess drained. In cases of doubt, intradural exploration should not be done until the presence of an abscess

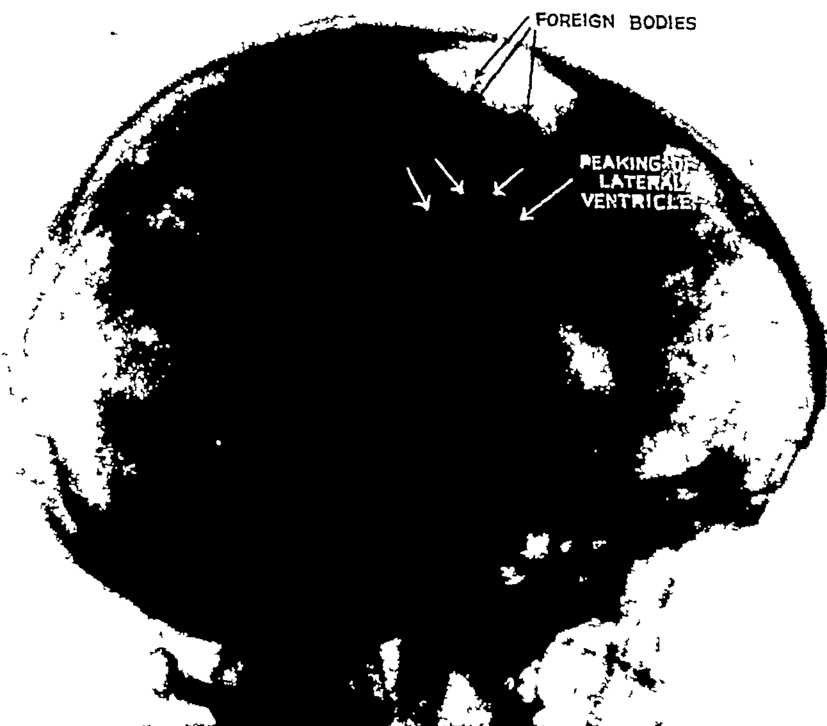


Fig 203

Small loose bony fragments in the depth of a wound commonly give rise to delayed infective complications Encephalography here proved that an intradural abscess was not present Peaking of the lateral ventricle is evidence of a meningo-cerebral scar

has been demonstrated by encephalography. On two occasions I have discovered an abscess embedded within the layers of the dura. The associated fistula in both cases refused to heal until the abscess was excised in one case and in the other widely opened and adequately drained (Fig. 204)

Spreading Osteomyelitis of Subacute or Chronic Type.—An indolent osteomyelitis may, as the result of thrombophlebitic processes, travel extensively along the diploic spaces. At one point the outer table may necrose ; here an external fistula may form At another point the inner table may sequestrate ; here an extradural abscess may form. Finally the whole segment of

the affected bone takes on the classical worm-eaten appearance, the X ray showing a fluffy area of irregular rarefaction enclosing dense sequestra.

Treatment is difficult because —

(i) Large areas of bone are affected

(ii) It is impossible to know by clinical or radiographic means just how far the inflammatory processes extend



Fig 401

Here an abscess enclosed within the layers of the dura mater was the cause of a persistent sinus on the top of the head. The X rays show the edges of the calvarial defect to be healthy; moreover there are no loose radio-opaque bodies in the depth of the wound. Encephalography proved that an intracerebral abscess was not present, as shown by the normal outline and position of the roof of the lateral ventricle

(iii) The overlying skin is unhealthy, it is riddled by multiple sinuses and in places may be adherent to the bone

(iv) The bacteria concerned are tenacious, or the tissues have a predisposition or lack of resistance to the organism concerned. Such conditions are very similar to tubercular infections of bone in their persistency

Theoretically the best treatment is complete removal of the

affected segment by block resection through healthy bone. The objections to this method are :—

(i) A large defect in the skull would be the result.
(ii) The danger that a large skin flap necessary for the exposure would not heal.
(iii) The difficulty in knowing how much bone to resect in order to cut beyond the boundaries of the infection
(iv) The danger that infection may start in the edge of the cut bone, however wide a resection be made.

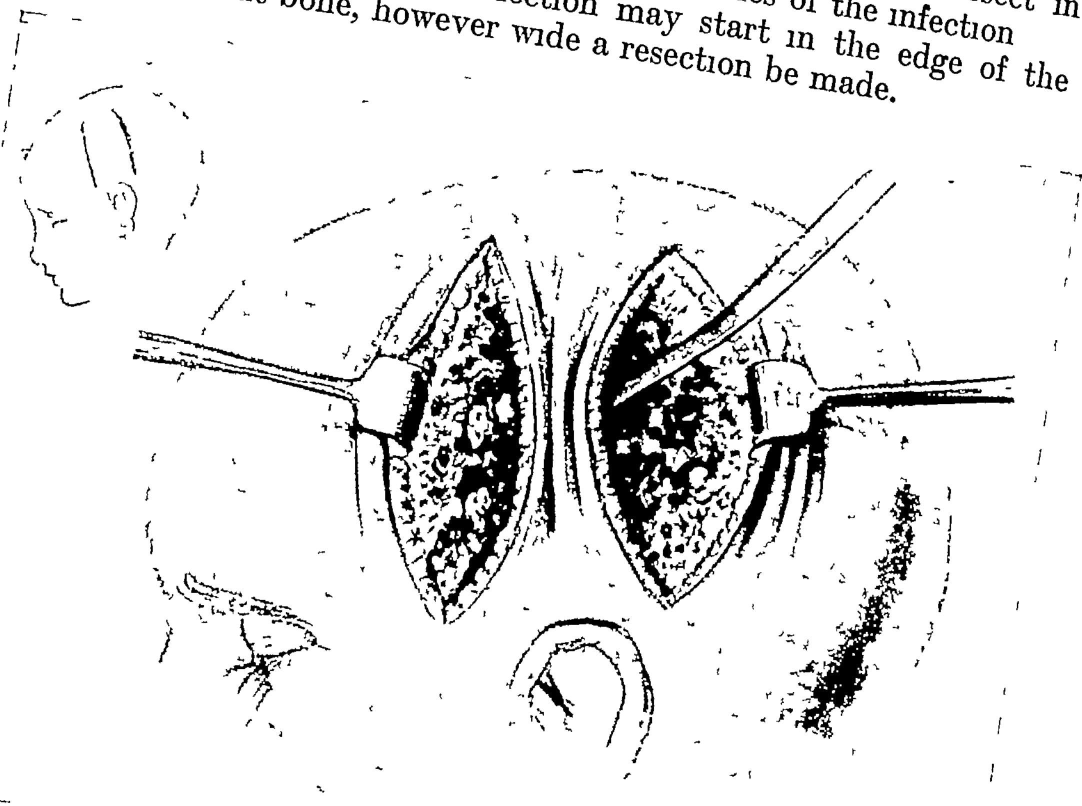


Fig 205
The surgical approach to spreading osteomyelitis of subacute or chronic type

In extensive infections it is probably wiser to approach the affected bone by means of a series of vertical parallel cuts through the scalp and to undermine the rectangles of skin so formed. In this way an unlimited area of the skull can be exposed without the danger of the skin retracting in the same way as when a flap fails to heal. The whole of the external table is then removed over infected diploe ; granulation tissue is scraped away and necrosed areas of the inner table excised The wound is then packed with sulphapyridine ; later a course of sunlight or deep X-ray therapy is applied to the head. Medical measures to bolster up the general health are also important. In fact, this is the type of infection which often necessitates treatment in a sanatorium.

CHEMOTHERAPY

In the acute and subacute varieties of osteomyelitis of the skull chemotherapy will, in many cases, be sufficient to cure the

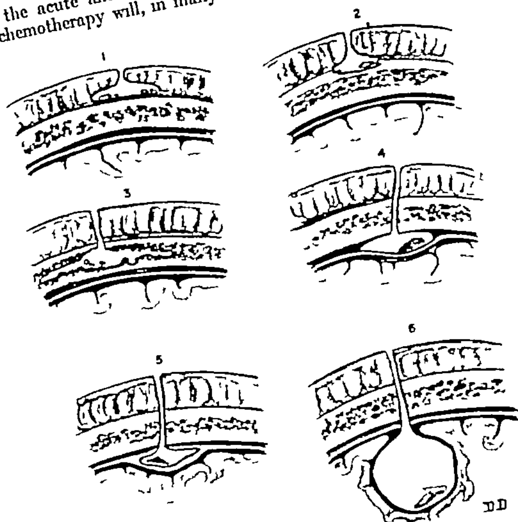


Fig. 406

The causes of persistent infection about the dome of the head.

1. Foreign bodies in the soft tissues.
2. Superficial osteomyelitis.
3. Cancellous osteitis.
4. Extradural abscess.
5. Intradural abscess.
6. Intracerebral abscess.

infection without the aid of surgery. If however pus forms it must be evacuated by surgical means and penicillin or streptomycin then instilled into the abscess cavity. It must be clearly understood that chemotherapy does not overcome the mechanical problems of cerebral compression which arise, for example when pus collects in the extradural or subdural spaces.

In cases of chronic osteomyelitis of the skull in which indolent fistulæ have formed, surgical removal of sequestra or foreign bodies is necessary as well as chemotherapy, whether this be administered systemically or locally. Local instillation of an antibiotic is of no value if the mechanical conditions are such that the drug cannot be brought into direct contact with the infected tissues. The object of surgical intervention is to procure amongst other things the most favourable conditions for the action of antibiotics applied locally (Fig. 206).

COMPLICATIONS

These are: (i) extradural abscess, (ii) meningitis, (iii) cerebral thrombophlebitis, (iv) encephalitis and (v) cerebral abscess.

Any of these conditions may result from a severe compound fracture or what appears to be a simple wound of the scalp. In the latter case the typical sequence of events is as follows: A child receives a simple blow on the head, say with a piece of slate, which lacerates the scalp. This is stitched or dressed and the child allowed to go home Within twenty-four hours he complains of headaches and soon is seriously ill with some kind of intracerebral complication.

Early treatment is essential if the child is not to die. Chemotherapy is, of course, started at once by mouth, but surgical intervention is also necessary. The wound should be reopened and the dura exposed by removing a piece of bone with a large trephine. If from the appearance of the dura, *i.e.*, by thickening, granulation tissue or a fistula, the route of infection can be determined, the dura should be opened when pus may be released. If a negative exploration results, no harm will be done if the wound is packed with sulphapyridine.

The treatment of cerebral abscess depends on whether or not the abscess is attached to the dura. When attached to the dura the abscess should be opened and drained. When subcortical in position, it should be aspirated as often as is necessary, then excised at a later date.

CHAPTER VIII

THE RESULTS OF INJURY TO SPECIAL PARTS OF THE BRAIN AND SKULL

THE VISUAL PATHWAYS

The Eyes—Rupture of the globe of the eye, as a result of cranial injury of the closed type, is almost unknown. In my clinical series of 1,000 cases it did not occur on a single occasion, which is a striking commentary on nature's skill in protecting important organs. No doubt this immunity of the eye from serious injury under peace-time conditions is due to (1) the protection it receives from the thick bony rim of the orbit, (2) the relative smallness of the eye which allows it to move away from injury whenever the orbital cavity is deformed, (3) the mode of accident, flat rather than pointed objects being responsible for the violence.

In road and industrial accidents, however, hæmorrhages into the globe of the eye are by no means uncommon, and bleeding may occur (1) into the anterior chamber, (2) into the vitreous, (3) into the retina or (4) between the coats of the eye.[1] Subhyaloid hæmorrhages may also occur, but these are usually associated with subarachnoid bleeding and are thought to be caused by blood being forced along the optic sheath from the intracranial cavity.

In air raid casualties penetrating wounds of the eye unfortunately are common, and are due either to bomb fragments or to small bodies, such as spicules of glass being blown forcibly through the air. No doubt they will prove to be common on the battle-fields (Fig 207).

Displacement of the axis of the eye occurs when the orbit is deformed by fractures involving the face or forehead. As it is the orbital floor rather than the roof which breaks, the eye is usually displaced downwards. Outward and downward displacements are often associated (Fig 208) but upward and inward displacements of the eye are exceedingly rare. Diplopia, however,

[1] Special number on "War Wounds of Eye and Orbit" *Post-grad Med Jour*, 1940 16.



Fig 207
Rupture of the globe of the eye is usually due to penetrating wounds In this case the distal sight of a rifle was blown through the orbit to become lodged in the base of the skull The eye was completely disintegrated.

is by no means an invariable sequel of displacements of the eye as is the case when ocular neuromechanisms are impaired.



Fig 208
Downward and outward displacement of the eye consequent on fracture of the orbit This man suffered from persistent diplopia

Since treatment of penetrating wounds of the eye is the concern of an ophthalmologist, nothing further need be said on this subject here save that treatment is urgent and no time should be lost before skilled assistance is sought.

The disability of temporary diplopia may be avoided by covering the affected eye with a shade, but as this interferes with binocular vision such occupations as the driving of cars or the working of a machine in a factory should be suspended. Persistent diplopia, due to displacement, which resists orthoptic treatment, may necessitate reconstruction of the orbit and replacement of the

eye before the visual images can be satisfactorily fused Such operations, of course, are best done by plastic surgeons

The Optic Nerves.—Gunshot wounds of the orbit may bruise or sever one or both optic nerves, producing blindness or defects in the visual field of a pattern that can readily be explained by the circumstances of penetration

In closed injuries, on the other hand the mechanism of optic nerve damage is not so obvious, and more will have to be said on this subject. In my series of acute head injuries the incidence of post traumatic blindness was about 0 5 per cent , and in 500 cases examined for litigation purposes it was found only in seven In Russell's[1] series of 600 cases, evidence of injury to the optic nerve occurred eight times. In Turner's series it occurred twenty five times in 1,550 cases[2] —

TURNER'S TABLE

Analysis of Cranial Nerve Injuries in a Series of 1,550 Cases

Olfactory	119
Optic nerve	25
Optic chiasm	3
Oculo-motor	15
Trochlear	15
Trigeminal (or its major branches, excluding supraorbital and infraorbital involvement)	3
Abducens	15
Facial	46
Vagus	1

It is rather surprising that damage to the optic nerve is so infrequent particularly as cranial injuries are so commonly basal in position As Rawlings[3] has shown the infrequency of optic nerve damage is possibly due to the fact that frontal fractures which converge on the pituitary fossa cross the sphenoidal fissure rather than the optic canal This explanation, however, is not entirely satisfactory because in Vance's[4] series of 512 necropsies, the optic canal was fractured in 10 per cent of cases, and in 61 per cent of von Hoelder's series of eighty eight basal fractures A fractured optic canal, of course, does not necessarily mean

[1] Russell, W R. "Injury to Cranial Nerves including the Optic Nerves and Chiasma." Brock's Injuries of the Skull, Brain and Spinal Cord," chap. v Baillière Tindall & Cox. London, 1943.

[2] Turner J W A. "Indirect Injuries of the Optic Nerve" *Brain*, 1943, 66, 140

[3] Rawlings, L B. "Surgery of the Skull and Brain." London, 1912

[4] Vance B M. "Fractures of the Skull." *Arch. Surg.*, 1927 14, 1023.

that the optic nerve has been contused, since it is displacement of fractured surfaces that causes injury to soft parts, and this does not occur in every case. Moreover, detachments of the anterior clinoid processes are usually associated with fatal degrees of violence. It may be presumed, therefore, that optic nerve injury would be more common if cases of the severer types of injury survived.

The actual cause of blindness is a subject of great controversy. Lindsay Rea [1] inclines to the view that it is due to blood within the optic nerve sheath, and although he did not actually make the statement he presumably means by compression. At autopsy, intravaginal bleeding is certainly a common finding, and the extravasated blood may come from a subarachnoid hæmorrhage or from rupture of the retinal arteries and veins which cross to the nerve from its sheath. Against this view is the non-occurrence of optic nerve blindness in cases of subarachnoid hæmorrhages caused by spontaneous rupture of a congenital aneurysm of the circle of Willis. When blindness does occur in such cases it is due to a subhyaloid hæmorrhage, which can be seen on ophthalmoscopy, and not to compression of the nerve fibres. Traquair believes that rupture of the small vessels supplying the nerve explains its loss of function. This may explain some but by no means all cases of blindness. Stretching of the nerve fibres is another possible mode of damage, but this mechanism must be rare, otherwise avulsion of the nerve would occur more frequently than it does.

Cone [2] has shown by histological methods that blindness may be caused by contusion of the nerve within the optic canal, and as will be shown later, this is in my experience an important mechanism of injury.

Clinical findings are variable and depend on degree and extent of injury and on the presence or absence of lesions in neighbouring structures, such as the oculomotor and sympathetic nerves. The eye may be completely blind and the direct light reflex absent. In spite of this, the pupil is not necessarily dilated, since it may be kept contracted by the consensual reflex. An appreciation of light or of hand movements may be all that is left of visual acuity; in these cases the direct light reflex is present though sluggish. Incomplete injuries produce all types of visual field defects. For example, central vision may be spared or absent, and peripheral loss may be quadrantic or scotomatous.

Tabulated below are the main findings in seven cases of blindness following head injury. In six cases the blindness was unilateral, but in the bilateral case, which suggested injury of the optic chiasma, the presence of fractures in both orbits was against this

[1] Lindsay Rea, R "Neuro-ophthalmology" Heinemann Ltd London, 1938

[2] Cone, W V. Personal communication

view A fracture of the optic canal was demonstrable by radiography in three only of the seven cases, although fracture of the anterior fossa or orbit was present in six. In six cases the optic disc was pale, with clean-cut edges typical of primary atrophy, and in one only was the disc normal in colour No doubt the absence of atrophy in this latter case can be explained by the fact that the disc was seen only three days after injury, three weeks later this disc also showed signs of atrophy In the six unilateral cases the blood vessels of the disc were found to be normal on comparison with those of the opposite side, meaning that the arterial supply of the retina was unimpaired The veins, moreover, were not engorged nor the disc œdematous, as would be expected if the venous return of the retina had been obstructed by an intravaginal hæmorrhage In one case that came to autopsy the nerve fibres had been compressed in the optic canal and there was an unmistakable indentation at the point where this had occurred

TABLE

Age	Sex	Nature of Injury	Severity of Injury	Side	Visual Acuity	Size of Pupil	Direct Light Reflex	Consensual Light Reflex	Discs	Vessels of Discs	Prognosis	Fracture of Frontal Bone	Fracture of Optic Foramen
[illegible]	M	[illegible]	[illegible]	L	[illegible]	[illegible]	Sluggish		Atrophic	Normal	Bad		
[illegible]	M	[illegible]	[illegible]	L					Normal then atrophic				
[illegible]	M	[illegible]	[illegible]	L					Atrophic				
[illegible]	M	[illegible]		R	[illegible]		Absent						
43	M	[illegible]	[illegible]	L	[illegible]	Fully dilated	Sluggish						
12	F	[illegible]	[illegible]	R		Fully dilated	Absent						
[illegible]	M	[illegible]	[illegible]	L & R	[illegible]	[illegible]	[illegible]			[illegible]			

To give an opinion on prognosis of optic nerve blindness in the early stages is difficult Perimetry may be helpful. If a field defect shades at its periphery, the sight in the shading field may recover because traumatic œdema and not irremediable structural injury may be the cause Theoretically, treatment of optic nerve blindness should be immediate exposure of the nerve at the clinoid process and decompression of the optic canal In practice of course this is never done because an accurate diagnosis cannot be made as long as the patient is unconscious, and when blindness is discovered it is usually too late for treatment to be of any avail Moreover, there is no guarantee that early operation will accomplish anything useful, the injury being in the nature of a contusion rather than of a compression

"Delayed post-traumatic blindness" has occasionally been described. It may result from strangling of the nerve by fibrous



Fig 209
The right optic canal, as shown by radiography

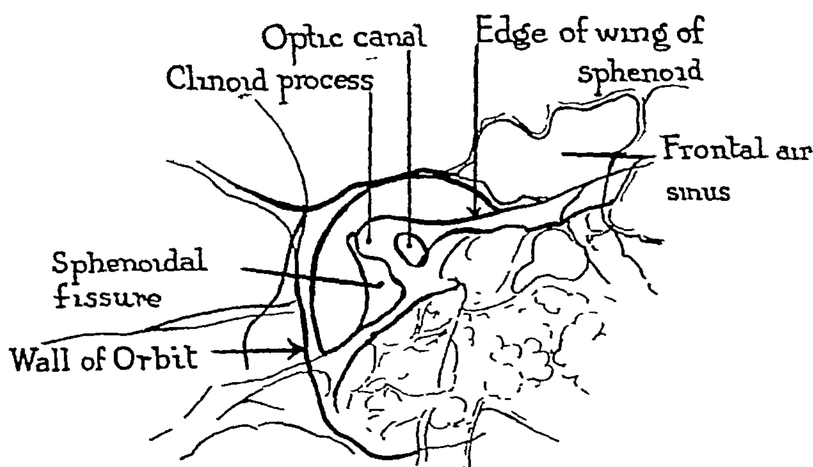


Fig 210
Key to Fig 209

tissue or callus as a fractured optic canal heals or from a progressive arachnoiditis. Operative treatment is always indicated in these cases and consists of removal of the roof of the optic canal after the floor of the anterior fossa has been exposed through an osteoplastic flap (Figs 209-212).

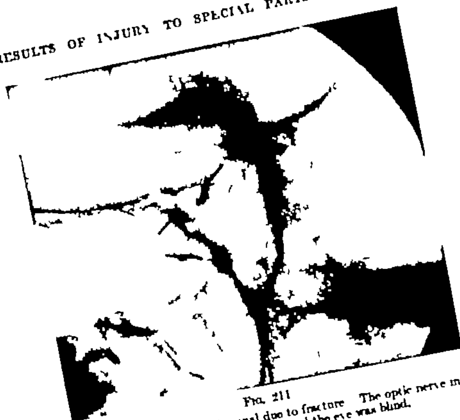


FIG. 211

Narrowing of the left optic canal due to fracture. The optic nerve in this case had been contused and the eye was blind.

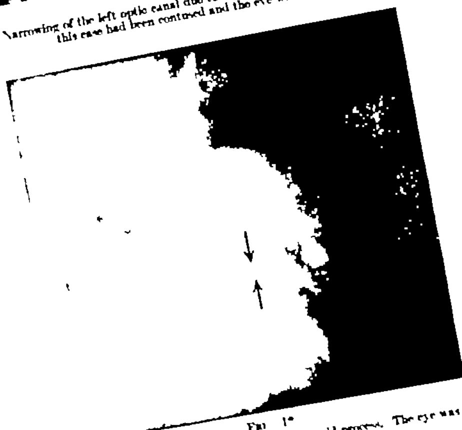


FIG. 212

Fracture of the base of the left anterior clinoid process. The eye was blind on this side.

The Intracranial Pathways (Fig. 213).—It is surprising how rarely the intracranial pathways are damaged in view of their

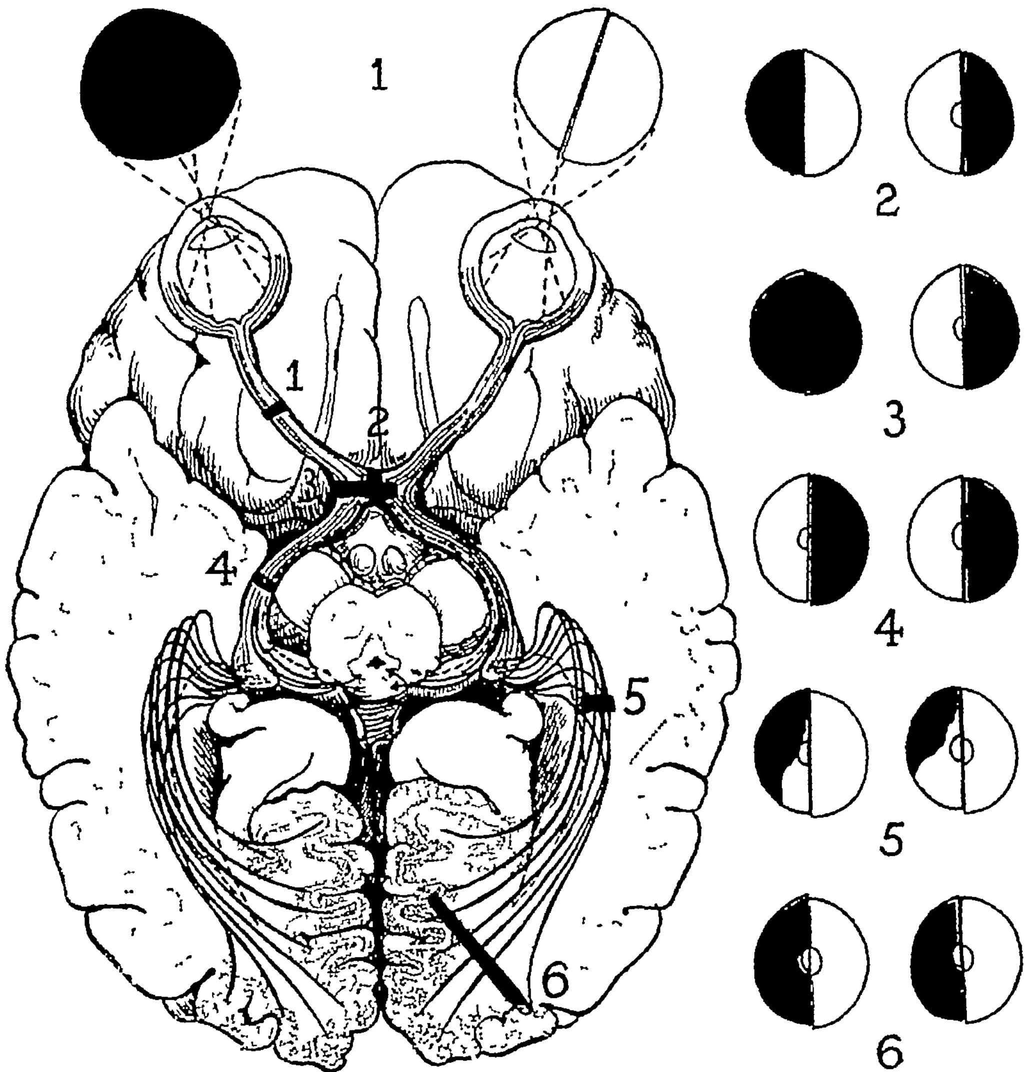


Fig 213

Injuries of the visual pathways

1, Injury to the optic nerve produces unilateral blindness
2 and 3, Injuries of the optic chiasma may produce bitemporal hemianopia or complete blindness in one eye and blindness in the other temporal field
4, Injury of the optic tract produces homonymous hemianopia of the congruous type with loss of half-macular vision
5, Injury to the optic radiations produces homonymous hemianopia of the incongruous type
6, Injury to the poles of the occipital lobes produces homonymous hemianopia of the congruous type, usually with sparing of macular vision, since macular vision is widely represented in the occipital lobe or is anteriorly placed

extent and of the exposed position of the optic tracts as they lie at the base of the brain.

The optic chiasma is sometimes affected, but rarely by contusion. Loss of vision in these cases, as Traquair, Dott and

Russell[1] have shown is due to rupture of the small chiasmal vessels with resulting ischaemia of the nerve fibres The most characteristic defect is a bilateral loss in the temporal fields of vision (bitemporal hemianopia) or a total blindness in one eye and a temporal loss in the other

Damage to an optic tract is a very rare injury indeed and shows as a congruous homonymous hemianopia

Lacerations of the occipital lobes or of the optic radiations, with resulting loss of vision in the contralateral visual fields, are sometimes caused by compound depressed fractures or perforating wounds When the occipital lobe is concerned (Fig 214), the

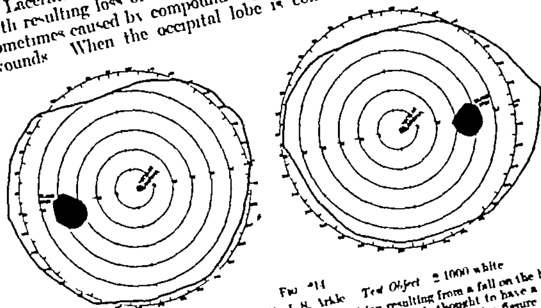


Fig 214 Test Object 2 1000 white

Name Dr M Perimetrist Dr J R Arkle

A homonymous scotoma involving central or macular vision resulting from a fall on the back of the head. This is a very unusual finding since macular vision is thought to have a very wide representation in the visual cortex or to lie in the region of the calcarine fissure itself and not at the tip of the occipital lobe

pattern of the visual loss is symmetrical in both fields (congruous homonymous hemianopia) whereas in injuries of the optic radiations the loss in each field is asymmetrical (incongruous homonymous hemianopia).

INJURIES OF THE CRANIAL NERVES

Sense of Smell.—Loss of smell follows injury of the head in about 5 per cent of cases and is particularly liable to occur when an injuring force has been applied to the head anteriorly or through the bones of the upper part of the face In itself it is of no great importance except in those people whose occupations are dependent to some extent on their sense of smell

It is, however a serious disability when the power of

[1] Traquair H M Dott N M and Russell W R "Traumatic Lesion of the Optic Chiasma" Brain 1935, 58, 398

appreciating aromas of food or wines is lost, as this interferes with one of the primary pleasures of life. One of the peculiar findings in head injuries is that although loss of smell may apparently be complete, that of taste (other than of salt, sour, bitter, sweet) may not be impaired. Possibly the ambiguous findings in these cases may be explained by the fact that sense of smell is not completely absent but is so faint that a gaseous stimulus passing through the anterior nares cannot be appreciated, whereas it may be possible when it arises in the nasopharynx. In many cases of loss of smell it is possible to demonstrate a fracture of the anterior fossa by radiography In such cases where fracture or dislocation of the cribriform plate is not present the olfactory filaments have probably been torn as the brain moved across the base of the skull at the time of accident. Avulsion or tearing of the olfactory tract, described and illustrated by Cairns,[1] is occasionally seen at operation or at autopsy. Occasionally the olfactory centres may be contused and lead to uncinate attacks In one of my cases, epilepsy of the convulsive type following injury was preceded by a horrible smell the nature of which could not be described save that it was offensive and nauseating In such an uncinate attack I have seen a man spit out and shake his head in disgust at the nastiness and reality of the sensation (Fig 215).

Prognosis in anosmia is doubtful. Improvement to some extent is usual, but full recovery is rare when loss of smell has been complete. On no occasion have I known of recovery when complete loss of smell has persisted for more than three months

An important pronouncement has been made recently by Leigh[2,3] on the subject of olfactory injuries. In a series of 1,000 consecutive cases of head injury admitted to a military hospital the sense of smell was damaged in 72 of them (7 per cent.) In 41 cases there was complete anosmia ; in 31 cases there was partial anosmia Recovery of sense of smell occurred in 6 cases only, and this usually before six months had elapsed after injury. Apparently olfactory injury occurs irrespective of the site of application of the accidental violence to the head. It is important to know that impairment of sense of smell is usually associated with severe injuries of the closed type Of the 72 cases reviewed by Leigh, 26 were so seriously incapacitated that they had to be discharged from the Services. Taste was affected in only 6 cases, and then only when total anosmia was present.

A very useful guide in testing the veracity of a patient's statement as regards loss of sense of smell is that sensations dependent

[1] Cairns, H "Injuries of the Frontal and Ethmoidal Sinuses, with Special Reference to Cerebrospinal Rhinorrhœa and Aeroceles" *Jour Laryng and Otol*, 1937, 52, 589

[2] Leigh, A D "Defects of Smell after Head Injury" *Lancet*, 9th Jan 1943, p 38

[3] Ogle, W *Med-chir Trans*, 1870, 53, 263

on the integrity of the trigeminal nerve should be present if this nerve has not been damaged and such damage may readily be shown by loss of common sensation Therefore if a patient is analysing sensations carefully and giving accurate answers, he

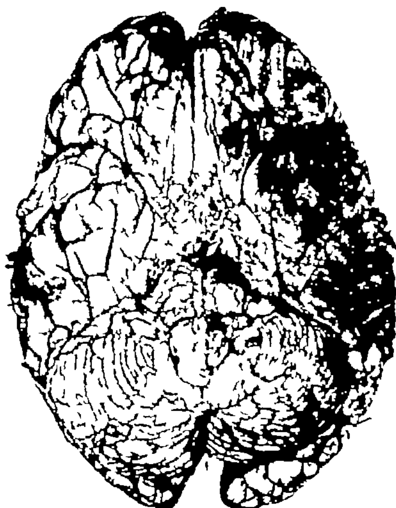


Fig. 215

A severe basal injury with extensive laceration of the under surface of the left temporal lobe Judging by the areas of maximum bruising the brain must have been travelling forwards and to the left in relation to the base of the skull at the time of injury On the left side the olfactory tract is bruised; on the right side the olfactory tract is completely severed. The uncal region is badly bruised on the left side

ought to be able to appreciate pungent vapours in the nose and to recognise salt or vinegar on the tongue The significance of these tests should never be allowed to come to the knowledge of the public otherwise they will lose much of their usefulness in helping medico-legal assessments to be made

The Oculomotor Mechanism.—Damage to the third, fourth and sixth cranial nerves in closed head injuries usually results either from fractures of the orbit or from stretching of the nerves in the posterior fossa as the brain stem is displaced at the moment of violence.[1] Penetrating wounds which damage the ocular nerves other than within the orbit usually cause fatal injuries by severing large intracranial vessels.

A complete paralysis of any ocular muscle supplied by the third or sixth nerve usually causes an obvious squint, and diagnosis of the affected muscle may be made with certainty (Fig. 216) On the other hand, when a muscle is merely paresed it is often impossible to detect malposition or even imperfect movement of the eye concerned. In these cases a careful analysis of the resulting diplopia is necessary if the affected muscle is to be discovered. Diagnosis depends on the fact that when an eye is made to look in the direction of pull of the weak muscle, true and false images become more widely separated Such separation is most easily recognised if the eyes are covered with different coloured glasses and a white light used for the object of fixation, so that false and true images are seen in different colours. Doubt is thrown on a patient's observations when he complains of the continuance of diplopia when an object is placed in the extreme temporal field of one eye and outside the nasal field of the other Such placing of the object of fixation is, of course, possible because the projection of the nose limits the nasal field of either eye, and this limitation can be mapped out by means of direct confrontation. Tests are carried out as follows: First, the patient's affected eye muscle is detected by alterations of the movements of the true and false images Then the patient is asked to look directly into the examiner's eyes and a small rounded object such as a white hatpin is held at an equal distance from the patient's and

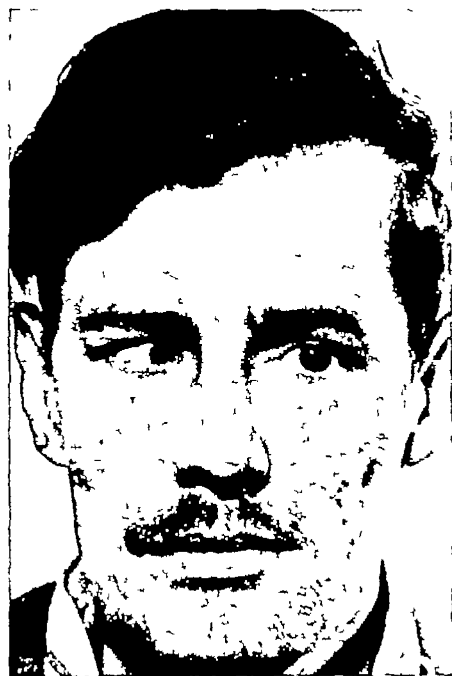


Fig 216
Paralysis of the external rectus muscle of the left eye The man is looking towards his left ear Complete recovery occurred three months after this photograph was taken

[1] Cross, A G "The Ocular Sequelæ of Head Injury" Lecture delivered at R C S E 30th December, 1947

examiner's eyes. The examiner now closes his eye which is opposite to the patient's faulty eye and keeps the other open. The patient is then asked to look at the head of the hatpin and the examiner does likewise. The hatpin is then moved towards the side of the closed eye until it just disappears from the nasal field of the open eye. If accurate observations are being made by the patient the complaint of diplopia should cease when the object of fixation passes out of range of the examiner's nasal field, as thus corresponds with the patient's nasal field of vision.

Diplopia, the result of damage of an ocular muscle or of contusions of the brain stem, usually recovers spontaneously within a few weeks or months. Persistent diplopia from any cause is exceedingly rare and necessitates treatment in an orthoptic clinic. When orthoptic treatment is not available the good eye should be covered with a shade for periods in the day and attempts made to move the other eye along the line of paralysis. A great deal of patience and understanding is necessary for this type of therapy.

Injuries of the Trigeminus.—Areas of anæsthesia about the head and face often are the result of severance of branches of the trigeminus by scalp wounds received at the time of accident. Most commonly the supra orbital and supratrochlear nerves are divided in the forehead just above the supra orbital notch. This leads to anæsthesia of the anterior two-thirds of the scalp on the same side. Contusion or laceration of one or more of the three divisions of the trigeminus or of its large branches may occur in osseous injuries as the nerves course through channels or openings in the bone to reach the face from the cranial cavity. For example, a fracture of the superior maxilla may lead to damage of the infra orbital nerve with resulting anæsthesia of the cheek (Fig. 217). Fractures of the lower jaw with displacement lead to severance of the dental nerve and numbness of the lower lip, teeth and jaw. In closed head injuries the Gasserian ganglion is occasionally injured by basal fractures that cross the tip of the petrous bone. Hæmorrhages into the ganglion sheath occasionally produce a condition which is indistinguishable from herpes zoster. The results of gunshot wounds are heterogeneous, and varying degrees of injury may be inflicted on the trigeminal nerve at any point from its posterior nerve root to its peripheral filaments.

Apart from incised and penetrating wounds, complete loss of sensation in any area of the face or scalp is rare. Usually *sensation* is partially and not completely destroyed; in these cases recovery of some degree may be expected, although normal sensation rarely returns.

It must be remembered that, subjectively, numbness is a very real and unpleasant sensation, the characteristic feature of which may be intense cold, burning, swelling or a creeping feeling under the skin. Occasionally an extremely painful interstitial trigeminal neuritis or paroxysmal neuralgia may be the result of an injury to the Gasserian ganglion.

Objectively, as sensation returns to an anæsthetic area, the skin may become hypersensitive and remain so for the rest of the patient's life. In medico-legal circles the question is often asked



FIG 217

An oblique shoot will demonstrate the lower margin of the orbit satisfactorily In the type of fracture shown the infra orbital nerve is always contused and occasionally severed

whether anæsthesia of the scalp is likely to lead to loss of or change of colour of the hair. In my experience of a series of 300 cases of nerve section or alcohol block done for trigeminal neuralgia this complication has never been observed.

The Syndrome of the Jugular Foramen.—The glossopharyngeal, vagus and spinal accessory nerves, in this order from before backwards, pass through the jugular foramen to enter the neck. On account of their protected position they are very rarely injured, and when this does occur the violence is usually overwhelming and leads to fatal results.

Injury to the Hypoglossal Nerve.—In closed head injuries I have never seen a case of contusion or laceration of the hypoglossal nerve alone. Usually injury to the hypoglossal nerve is associated with the syndrome of the jugular fossa, and this combination

has been described by Collet[1] following a penetrating wound of the posterior fossa and by Galand[2] in the case of a patient who fractured the base of the skull by diving into shallow water

INJURIES OF THE AUDITORY AND FACIAL NERVE

Deafness and Facial Paralysis.—As stated in Chapter I the body of the petrous bone offers stout resistance to injuring forces and deflects to its base most fractures which run towards it either from the middle or from the posterior fossa. This, no doubt, is the reason why fractures involve the middle ear far more frequently than the internal ear

Forces of great magnitude occasionally break the body of the petrous bone transversely and sever the seventh and eighth nerves as they lie within the internal auditory canal (Fig 218). Such injuries, however, are usually fatal and so are of no clinical importance.

In any case, fractures involving the middle ear are those with which the surgeon is chiefly concerned not only because they may result in deafness and facial paralysis but also because they may result in otitis media and meningitis, for which active treatment is possible. Fractures involving the middle ear may be divided into two distinct groups (1) those confined to the base and (2) those which are continuation fractures from the vault

Deafness.—Middle-ear deafness may result from either type and is caused by one or a combination of the following factors (1) blood in the tympanum, (2) dislocation of the ossicles, (3) blocking of the eustachian tube, (4) rupture of the membrana tympani, and (5) interference with the action of the tensor tympani. The membrana tympani is not invariably ruptured when bleeding has occurred into the middle ear and in such cases auroscopic examination will show the drum to be bulging and discoloured also bleeding into the middle ear can occur without fracture of the petromastoid bone

Diagnosis of the exact cause of deafness following head injuries is always difficult and is essentially a problem for an otologist

Injury to the head with or without demonstrable petromastoid damage is very serious in people who were losing their hearing before accident, since any pre-existing degenerative or chronic

[1] Collet, M. "Sur un Nouveau Syndrome Paralytique Pharyngo-Laryngé par Blessure de guerre" (Hemiplégie glosso-laryngo-scapulo-pharyngée) *Lyon Méd.*, 1915, 124, 1[illegible]

[2] Galand, G. "Syndrome total des Quatre Derniers Nerfs Craniens (Collet) avec Paralysie du Sympathique ou Syndrome de l'Espace Latéro-Post-rétro (Villaret)" *Ann. d'oto-laryng.* 193[illegible], 1[illegible]

inflammatory process affecting the ear may be considerably accelerated thereby.

Prognosis is always doubtful in middle-ear deafness and



Fig 218

Fracture of the body of the petrous bone, as shown in the above radiograph, is uncommon In this case there was deafness of the internal ear type and complete facial paralysis The man died of meningitis ten days after injury

definitely bad when the internal ear is affected [1] According to Davis,[2] if improvement in hearing does not begin within eight weeks following an accident the chances of recovery are small

Facial Paralysis.—In my experience facial paralysis has been more commonly due to fractures running into the base from the

[1] "Discussion on Injuries of the Ear" *Proc Roy Soc Med*, 1940, 34.
[2] Davis, E D D *Proc Roy Soc Med*, March 1931, 69

Fig. 219

This is the type of fracture which runs across the roof of the middle ear and produces facial paralysis.



Fig. 220

An oblique view will often demonstrate the type of fracture which runs from the vault downward across the roof of the middle ear



Fig 221

A rare combination of oculomotor and facial nerve paralysis



Fig 222

Two months after the injury there was evidence of recovery of the paralysis of the third nerve



Fig 223

A combination of right facial and right external rectus muscle palsy The child is attempting to look to the right Hearing was impaired on the right side



Fig 224

The man is looking to the left and is attempting to draw up both corners of the mouth Paralysis of the right facial and left external rectus muscles can be plainly seen Hearing was not impaired

vault than to those confined to the base In three cases, two at operation and one autopsy, I have been able to trace in detail the course of a fracture which led to paralysis On each occasion the fracture started in the squama of the temporal bone or in the posterior inferior angle of the parietal bone (Figs 219 and 220), and then ran downwards into the base of the mastoid bone just behind the posterior wall of the external auditory canal At this point it turned inwards across the roof of the middle ear and ended in the facial canal distal to the geniculate ganglion This,

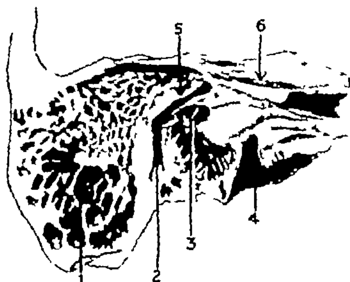


Fig 223

A cross-section of the petromastoid bone showing the facial canal.

1 Mastoid air cells. 2, Descending part of the facial canal.
3, The stapes. 4 The carotid canal.
5, The point at which fracture of facial canal usually occurs.
6, Groove containing superior petrosal nerve

I believe is the typical fracture which leads to damage of the facial nerve

In nearly all cases there is bleeding into the external auditory canal on the affected side On the other hand, leaking of cerebrospinal fluid is relatively rare There may or may not be frank signs referable to the labyrinth or cerebellum

Other cranial nerves are occasionally involved (Figs 221–224)

Facial paralysis may be immediate or delayed

Immediate—Immediate facial paralysis is the result of contusion or compression of the facial nerve in the transverse part of the facial canal

Early Delayed—Paralysis occurring a few days after injury may follow infection (Fig. 226) or may be due to the accumulation of blood clot around the nerve or of œdematous swelling of the nerve fibres themselves.

Late Delayed.—Paralysis developing after many months or a year and not due to infection may occur, and is due to constriction of the nerve fibres by the cicatrisation of healing processes

Prognosis of Facial Paralysis.—Prognosis is difficult to predict in the early stages of paralysis as little guidance can be obtained from scientific data However, at least 90 per-cent. of cases recover, and in my experience one case only in thirty has failed to do so In this case failure was due to fracture of the internal auditory and not of the facial canal, and deafness of the internal-ear type was also present. An incomplete or delayed paralysis is more favourable than a complete and immediate one, since the causative lesion is more likely to be amenable to surgical treatment if the paralysis does not resolve spontaneously Favourable cases show signs of improvement within two months and usually within two or three weeks (Figs. 227 and 228). There is little hope of recovery if no improvement is evident after a year.



Fig 226
Facial paralysis, the result of suppurative otitis media following a petromastoid injury

An incomplete injury of the facial nerve may later lead to facial spasm, a condition equally disabling as, and probably more disfiguring than, paralysis.

In many cases of facial nerve injury the chorda tympani nerve is injured, with consequent loss of trigeminal taste (salt, sour, bitter, sweet) on the same side of the tongue Renewal of conduction of taste impulses may be the first sign of recovery, as shown in two of my cases which were observed in detail. Sense of taste first returned and this was followed shortly by improvement in the facial paralysis. Another point of interest is that taste is lost on the same side of the tongue but retained on the same side of the palate. This is of anatomical rather than of prognostic

value, but it does show that the facial nerve is injured distal to the geniculate ganglion, otherwise the great superficial petrosal nerve, which is the gustatory pathway from the palate would also be injured

Treatment *Infection* —As described in the previous chapter bleeding from an external auditory meatus must always be regarded seriously and treated with great care, otherwise meningitis may result if the dura has been torn In fact, the condition should



FIG 227

An example of delayed facial paralysis coming on ten days after injury



FIG 228

Considerable improvement had taken place fourteen days after treatment was started

be treated with the same aseptic considerations as in every compound fracture of the skull The pinna should be carefully cleaned and covered with a sterile bandage The external auditory meatus should never be syringed nor any other method used forcibly to remove the blood clot Whenever a fracture opens into a middle ear which is already infected a mastoidectomy should be performed as soon as the patient's general condition will allow It is unwise to be conservative in these cases and to wait for complications such as spreading osteomyelitis meningitis or facial paralysis to develop before deciding to operate Also if infection develops in a primarily uninfected ear radical treatment is again indicated Sulphonamide or penicillin therapy as a prophylactic measure should be started early in all cases where a fracture is known to have involved the middle ear

Deafness —For deafness little active treatment is possible It

is, in fact, restricted to paracentesis for drainage of blood from the middle ear when the drum is intact and bulging. The danger of paracentesis is that infection may be introduced and otitis media result if complete aseptic precautions are not taken.

Facial Paralysis.—Theoretically, the facial canal should be decompressed in all cases of traumatic facial paralysis whether infection is present or not, particularly when paralysis develops after an interval. In the absence of infection, however, conservative measures give such a high percentage of good results that operative treatment in the early stages should be withheld. An exception may be made to this rule when the facial muscles are rapidly wasting and when electrical reactions of degeneration become evident.

A wire splint, enclosed in fine rubber tubing, crooked over the ear at one end and hooked into the corner of the mouth at the other, should be used immediately and continuously to keep paralysed muscles from being overstretched Massage and electrical stimulation should be employed every day until full recovery is attained, or until other forms of treatment are decided upon. After three months of conservative treatment, if there has been no sign of improvement, the facial canal should be explored and decompressed whatever the electrical reactions of the muscles may be. Adequate exploration of the facial canal necessitates radical mastoidectomy. When infection has complicated a fracture the fracture lines should be followed and excised as far as anatomical conditions will allow.

INJURIES TO THE BASAL NUCLEI

The hypothalamus [1, 2] consists of a collection of nuclei situated in the brain immediately above the sella turcica in the floor of the third ventricle. The nuclei may be divided into three groups: (1) anterior, including the paraventricular and supra-optic group; (2) the middle, including the tuber, lateral, dorsomedial and ventromedial hypothalamic nuclei; and (3) the posterior group, including the posterior hypothalamic nucleus and mamillary bodies. By means of connecting fibres all these nuclei are brought into close communication with each other and with the cortex and optic thalamus.

The hypothalamus is the part of the brain which is concerned in regulating the functions of the autonomic nervous system. In its posterior part is situated the centre for control of the sympathetic nervous system. Its central and anterior parts are concerned with parasympathetic regulations

[1] Le Gros Clark, W E, Beattie, J, Riddoch, G, and Dott, N M. "Hypothalamus." Oliver & Boyd Edin, 1938

[2] Fulton, J F "Physiology of the Nervous System" Oxford University Press, 1938

The various functions of the hypothalamus have been determined chiefly by animal experiment. Stimulation of the posterior hypothalamus produces changes characteristic of sympathetic hyperactivity, such as increase in the heart rate, rise in blood pressure, increased metabolism, dilatation of the pupil and inhibition of the intestinal movements. Destruction leads to the opposite effects including lethargy. The tuber is concerned with sweating and with movements of the stomach and intestines. Destruction of the paraventricular nucleus causes hypoglycaemia and destruction of the supra-optic nucleus diabetes insipidus. The whole of the metabolic processes of the body are, in fact, under control of the hypothalamus. Many observations made experimentally have been confirmed in man when the floor of the third ventricle has been accidentally bruised in the removal of pituitary tumours and when degenerations or neoplasms have affected these regions.

Injury to the hypothalamus resulting from a violence to the head is by no means rare as proved by autopsy findings. This is not surprising since fractures of the base tend to converge on the pituitary fossa immediately above which the hypothalamus is situated.

The most typical hypothalamic syndromes which occur as a result of head injuries are as follows:—

(i) Hyperthermia.
(ii) Diabetes insipidus.
(iii) Fröhlich's syndrome.
(iv) Acute erosions of the stomach.
(v) Diencephalic epilepsy.
(vi) Hypersomnia.

ULCERATION OF THE ŒSOPHAGUS AND GASTRO-INTESTINAL CANAL

The etiology of gastric and duodenal ulceration has long been a subject of clinical and experimental interest. Cushing[1] was the first to show that lesions in the region of the third ventricle or hypothalamus occasionally resulted in gastro-intestinal ulceration, and it is now becoming evident that hypothalamic influences are of considerable importance in the production of so-called nervous dyspepsias.[2, 3]

[1] Cushing, H. "Peptic Ulcers and the Interbrain." *Surg. Gyn. and Obst.*, 1932, 55, 1.
[2] Beattie, J. "Hypothalamic Mechanisms." *Canad. Med. Ass. Jour.*, 1932, 26, 400.
[3] Tedeschi, C. G. "... the Mucosal Lesion in Rat ... Submitted to Head Trauma." *Proc. Soc. for Exper. Biology and Medicine*, 1944, 57, 768.

ACUTE GASTRIC EROSIONS

Acute gastric erosions and perforations of the stomach and œsophagus of neurogenic origin certainly occur, and the following case will show that they may result from injury to the head. A youth under my care in the Stockport Infirmary died of acute peritonitis on the twelfth day after a severe injury to the head. It was ascertained from his mother that he had not previously suffered from dyspepsia. An autopsy examination, at which I was not present and which was not conducted by my friend Mr Andrew McGill, was made by an outside and inexperienced pathologist at the request of the coroner. He came to the conclusion that the youth had died of perforation of the lower end of the œsophagus and fundus of the stomach, consequent on corrosive poisoning. He did not attempt to explain the fact that the mouth and upper end of the œsophagus were not affected At the request of the hospital authorities I was asked to confirm the post-mortem findings. On reopening the abdomen I found definite evidence of recent inflammatory changes in the peritoneal cavity which could not be accounted for by post-mortem autolysis. The stomach and œsophagus were missing, as these had been removed and sent to the county analyst for examination of their contents. The brain had been sliced into many pieces and these were found tucked away in the pelvis. These were collected and fixed in a solution of 10 per cent formalin Later, with the aid of Professor Jefferson, the jigsaw puzzle of the brain was fitted together and petechial hæmorrhages were found in the floor of the third ventricle. The analyst's report returned negative for corrosive poisoning, and the boy's mother presented me with the specimen of the stomach and œsophagus. In this, perforated ulcers were found in the lower end of the œsophagus and upper end of the stomach (Fig. 229). Although histological examinations were not made of the walls of the ulcer, the specimen being inadvertently destroyed before this could be done, it is reasonable to presume that the gastric and œsophageal ulceration was neurogenic in origin and the result of the hæmorrhages in the hypothalamus. At no stage in his illness did the youth regain consciousness.

TRAUMATIC DIABETES INSIPIDUS

Traumatic diabetes insipidus usually declares itself early in convalescence, though it may not appear till several months after the injury. The outstanding symptom in this condition is insatiable thirst with a desire for water to assuage it. In severe

cases a patient will drink 1 or 2 pints of water at a time and has to take copious supplies to bed with him as he so often wakens in the night and wants to drink. Fluids taken by mouth are rapidly excreted by the kidneys so that a patient tends to lose weight rather than to become waterlogged. Severe headaches,

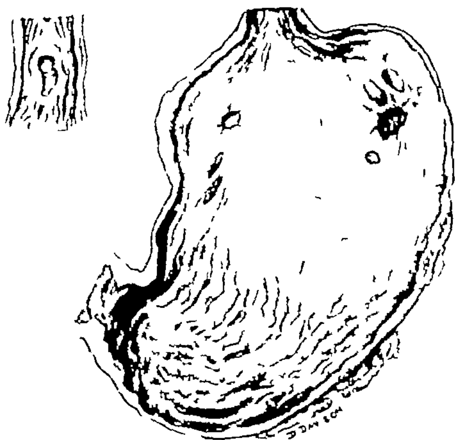


Fig. 20

Neurogenic ulcers of the œsophagus and fundus of the stomach resulting from hæmorrhage into the hypothalamus.

loss of physical energy and mental depression are commonly associated with the symptom of thirst. One young patient of mine who had been severely concussed and whose skull had been fractured through the pituitary fossa needed at least 12 pints of fluid in the day to satisfy his thirst, and once drank as many as 15 pints. The amount of water he passed in the night was so enormous that it seriously interfered with his sleep. This disturbance of water metabolism was readily controlled by hypodermic injections of 0·5 c.c. of pituitrin night and morning. In six months after the injury this young man made a complete recovery. The tendency in all cases of traumatic diabetes insipidus

is towards spontaneous cure. It is useless to restrict the amounts of fluids taken in an attempt to control the diabetes, because this only leads to dehydration and toxæmia Usually injections of pituitrin will keep a patient's thirst under reasonable control until spontaneous cure of the diabetes results. Severe and persistent cases may necessitate a total thyroidectomy as suggested by the work of Mahoney and Sheehan.[1]



FIG 230

A traumatic hæmorrhage into the basal ganglia Probably it is this type of injury which, in non-fatal cases, leads to signs and symptoms indistinguishable from those of infected processes involving the extra pyramidal ganglia (Parkinson's disease)

A pathological increase in weight due to excessive deposits of fat occasionally results from a basal injury. Hypersomnia following injury is extremely rare.

Apart from the above symptoms, which afford obvious evidence of damage to the hypothalamus, there are probably many others which also are the result of this type of injury, although they are usually attributed to anxiety or malingering. Frequently a patient complains of peculiar sensations of chilliness about the body and abdomen which might be vasomotor in origin. Diencephalic

[1] Mahoney, W, and Sheehan, D "Effect of Total Thyroidectomy upon Experimental Diabetes Insipidus in Dogs" *Amer Jour Physiol*, 1935, 112, 250

epilepsy [1] probably occurs more frequently than is usually believed For example, attacks of shivering and sweating for no apparent reason are probably hypothalamic in origin Emotional outbursts and surges of rage are occasionally seen in the acute stages of cerebral trauma, and these, it may with reason be suggested, are sometimes due to contusion of the optic thalamus

Whether injury to the caudate and lenticular nuclei can result in Parkinsonism [2] is a subject of great medico legal importance, and the problem has not yet been finally settled My own opinion is that it can, because a typical syndrome of the disease, which medical evidence can prove was not present before the accident, may be discovered in the early stages of convalescence following an injury Unlike the post encephalic form of the disease it is not usually a progressive pathological state (Fig 230)

The exact rôle played by the basal ganglia in the complication of acute cerebral trauma is at present obscure, but evidence is accumulating to show that it is an important one and worthy of more detailed investigation



Fig 231

Severe chemosis proptosis and intra and extra-ocular nerve palsies have occurred in this case of left-sided carotid-cavernous aneurysm. The right eye is already becoming affected.

PULSATING EXOPHTHALMOS

Although over 600 cases of pulsating exophthalmos have been described in the literature its incidence in any series of head injuries is exceedingly small. In the past fifteen years I have seen it on three occasions only and one of these was in America.

No doubt it is its dramatic appearance which accounts for so many cases having been described and certainly it is because of its distressing symptomatology that numerous bold surgical procedures have been used in an attempt to cure it (Fig 231) The underlying cause of the condition is a fistulous opening from the carotid artery into the cavernous sinus either directly or through an aneurysmal sac arterial blood being blown under relatively great pressure into the venous system of the cavernous sinus

[1] Penfield, W G "Diencephalic Autonomic Epilepsy" *Arch Neur and Psych* 1929 22, 358
[2] Weil, M. O., and Ousmansky V "Parkinsonisme Traumatique" *Rev Neurol* 1937 67 459

with the result that the tributary veins become enormously distended and pulsate with the heart beat.

Seventy-five per cent. of the cases of pulsating exophthalmos are due to trauma and 25 per cent. to spontaneous rupture of the artery. When the result of trauma, the majority are caused by closed fractures of the middle fossa and not by penetrating wounds.

The first symptom is a noise in the head which can be heard by the patient and on auscultation. In the early stages the noise may be present only in systole but later becomes continuous with a systolic exaggeration. In character the noise may be swishing, banging, beating or clanging, and is worse when the patient is lying down in a quiet room.

Usually the second symptom is diplopia consequent on stretching of the ocular nerves as they pass along the wall of the cavernous sinus. This is followed by protrusion and pulsation of the eye. According to Dandy,[1] this occurs within twenty-four hours in 33 per cent. of cases.

As the veins of the orbit distend, the eyeball is displaced according to the position of the main ophthalmic veins, and as these are usually situated in the upper and inner quadrant of the orbit the displacement is commonly downwards and outwards. Also, the conjunctiva become chemosed, and in serious cases the cornea may ulcerate. Papillitis and blindness are later complications.

The following case will illustrate the typical sequence of events in the life-history of a case of pulsating exophthalmos and how the condition may be cured by operative means

A young Australian soldier was enjoying himself one night in his billet with his friends, and on the way back to his room tripped and fell. He was concussed for a few hours and the next morning was aware of a feeling of fullness in the head. Some days afterwards he was conscious of a noise in his head and later was examined by my colleague Professor Nattrass, who made a diagnosis of a traumatic aneurysm of the internal carotid artery. When transferred to my care his only complaint was of an intermittent throbbing noise in the head. On auscultation over the anterior part of the temporal region on both sides a murmur could be heard which was synchronous with the heart beat and which resembled the clumping of heavy boots on the wooden floor of a large empty hall. Digital compression of the carotid vessels in the neck on the right side caused the murmur to disappear, but not when the vessels on the left side were compressed. At this stage there was a little fullness of the right upper eyelid but

[1] Dandy, W E "Carotid-cavernous Aneurysms (Pulsating Exophthalmos)" *Zentralbl. f Neurochir*, 1937, 2, 77 and 165

no pulsation in the orbital veins The eyeball was not displaced and did not pulsate. Compression of the carotid vessels for more than a minute caused a feeling of giddiness, thus proving that the collateral circulation and the circle of Willis was inadequate

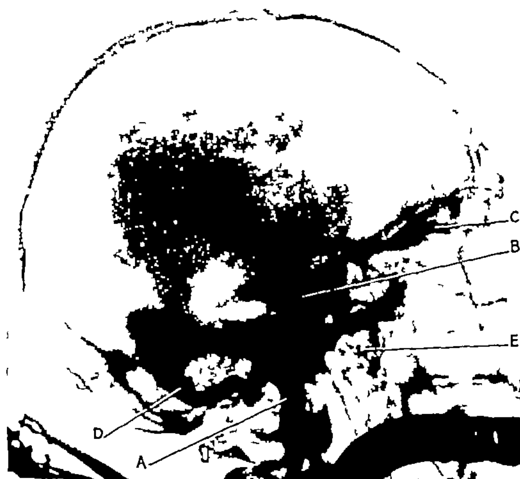


FIG 23.

An angiogram depicting the state of affairs in the case of a cavernous arteriovenous aneurysm due to trauma. The internal carotid artery A is seen coursing upwards through the neck to end in the cavernous sinus B Normally the S-shaped outline of the intracranial part of the internal carotid shows quite clearly before it breaks up into the branches of the anterior and middle cerebral arteries (see Fig 1). From the cavernous sinus the thorotrast has flowed into the ophthalmic vein C, the lateral sinus D and the pterygoid plexus of veins E. It is pulsation and engorgement of the ophthalmic vein which leads to pulsating exophthalmos.

to permit arterialisation of both sides of the brain through one carotid artery

It was decided therefore, to compress the vessels digitally in the neck for increasingly lengthening periods until occlusion of the vessels could be maintained for one hour without the development of symptoms

Whilst this treatment was in progress the patient developed

diplopia, but no obvious paralysis of the muscle could be demonstrated. On this evidence of retrogression it was decided to ligature his internal carotid artery in the neck On the morning of the operation his double vision was much worse, and there was a definite protrusion but no pulsation of the eyeball. The man felt ill and was very worried about himself.

Under intravenous Pentothal anæsthesia the internal carotid artery was exposed and an angiogram made by injecting 10 c.c. of thorotrast into the artery.

The X-ray showed that the shadow of the internal carotid artery ceased at the cavernous sinus and that the ophthalmic veins and lateral sinus were clearly filled with thorotrast (Fig. 232). This picture, of course, is what might be expected in view of the pathology of the condition.

The man was then allowed to come round from the anæsthetic until he was conscious and fully co-operative. At this stage I compressed the internal carotid artery between my finger and thumb for twenty minutes and repeated tests as regards muscle power were made on the opposite side of the patient's body. As no neurological signs developed the vessel was ligated firmly with stout silk and the wound left open for another fifteen minutes to make sure that hemiplegia did not develop

The noise in the head ceased at the moment of compression, and the man's mental attitude changed dramatically to one of optimism. For a month after operation the patient was left in bed with his head low and with the foot of the bed raised for the first two weeks.

CHAPTER IX

THE SEQUELS OF INJURIES OF THE HEAD

OF the people killed every year on the British roads—and before the war the number was rapidly approaching 9,000—at least 80 per cent. die as a result of injuries to the head, as judged by statistics of traumatic deaths in my own hospitals Furthermore, for each fatality due to cerebral causes there are five non fatal but severe injuries of the head which lead to prolonged morbidity and occasionally to partial or complete incapacity

In this chapter we are concerned only with the late results of cranial and cerebral injury, whether originally of the open or closed type and whether received under war time conditions or by accident in civilian life That these late results or sequels are multitudinous in kind is not surprising since the mechanism of most injuries to the head is such that the whole brain is subjected to the injuring force and suffers accordingly Neurones or nerve cells, neuroglia or interstitial tissue blood vessels and their controlling mechanisms, meninges and cerebrospinal apparatus are all damaged to some extent, as well as the scalp and the skull

The most common sequels which follow severe trauma can be classified thus —

I Of the Brain.

(a) *Those due to Diffuse Injuries*

Common Symptoms

1 Headaches
2 Dizziness
3 Insomnia.
4 Changes in disposition
5 Mental sluggishness in adults.
6 Intellectual retardment in children.
7 Psychoneurosis (anxiety neurosis)

Rare Symptoms

8 Hysteria.
9 Psychosis

(*b*) *Those due to Local Destruction of Brain Tissue by Contusions or Lacerations.*

1. Spastic paralysis.
2. Aphasia—motor and sensory.
3. Hemianopia.
4. Epilepsy—focal and general.
5. Sensory loss of discriminative type
6. Diplopia due to injury of the ocular nuclei within the brain stem
7. Intellectual impairment due to bilateral contusion of the frontal lobes
8. Diabetes insipidus, adiposis genitalis and ulcers of the stomach due to injuries of the hypothalamus.
9. Hydrocephalus
10. Delayed apoplexy.

II. Of the Cranial Nerves.

1. Loss of smell and taste.
2. Loss of vision.
3. Diplopia.
4. Sensory loss in the trigeminal field.
5. Facial paralysis.
6. Deafness due to contusion of eighth nerve.
7. Nerves nine to twelve are rarely damaged.

III. Of the Skull.

(*a*) *Closed Injuries*

1. Compression of brain due to depressed fragments.
2. Dizziness due to involvement of the labyrinth.
3. Deafness—of middle ear type.
   Deafness—of internal ear type.
4. Meningocele

(*b*) *Compound Injuries.*

1. Same as in closed type.
2. Infection:
   Chronic periostitis.
   Chronic osteomyelitis.
   Subdural abscess.
   Meningitis—focal or generalised.
   Localised intracerebral abscess.
3. Defects in the skull
4. Cerebral fungus.
5. Cerebrospinal fluid fistula—ear, nose, vault.

IV Of the Scalp

1 Loss of tissue with deformity
2 Loss of hair
3 Neuralgia.

THE POST-CONCUSSIONAL SYNDROME

In the recovery from any severe injury of the head there are three distinct phases which can be easily recognised and recorded

The first phase is the return of consciousness, which may take any time from a few moments to several weeks the usual period being a matter of days Secondly, there is a period of convalescence lasting about three weeks, at the end of which time the patient is able to get up and about. Following this there is a long process of gradual recovery before the patient attains his pre accident state This third phase is the period of the post concussional syndrome, and few escape without suffering to a lesser or greater extent from one or a combination of the following conditions pains in the head dizziness, insomnia, diplopia, dispositional changes or intellectual impairment

In the past few years I have examined and taken detailed notes of 500 cases of people suffering from the late effects of injury to the head, moreover I have had opportunity of comparing my findings with those of the patients' general practitioners and of other skilled observers An analysis of the main symptoms of this series of cases will be found in the following table —

ANALYSIS OF SYMPTOMS IN SERIES OF FIVE HUNDRED CASES OF POST-CONCUSSIONAL SYNDROME

Fault Vision		Deafness.	Loss of Smell	Loss of Taste.		Vomiting	Anaesthesia of some Part of the Body	Miscellaneous Symptoms.	Headaches.	
Unilateral Blindness.	General Deterioration of Vision			Partial.	Complete.				Bilateral.	Unilateral.
[illegible]	71	24	27	11	8	11	23	207	[illegible]	152
·6	1	4 8 %	5 4	2 2 %	1 6	2 2 %	4 6 %	41·4 %	59	30 4

Dizziness		Insomnia.	Bad Dreams.	Changes in Disposition.	Nervousness.	Unsteady Gait.	Insanity		Epilepsy
Labyrinth.	Blackouts.						Temporary	Permanent.	
18	88	[illegible]	84	[illegible]	215	171	18	15	30
3 6	17 6	[illegible]	16 8	[illegible]	43 0 %	34 2 %	3 6	3 0	6 0

1 Pains in the Head [1,2]—Headaches may be unilateral or bilateral they may come on suddenly and disappear equally

[1] Northfield, D. W. C. "Some Observations on Headache." *Brain* 193 **61**, 133.
[2] Schumacher, G. A., and Wolff, H. G. "Experimental Studies on Headache." *Arch Neur and Psych* 1941 **45**, 199.

suddenly, or they may develop slowly, come to a climax and then recede slowly; they may occur at some particular period of the day; and they may be precipitated or aggravated by exertion, excitement or worry. A common complaint is of a persistent and generalised discomfort in the head, associated with severe stabs of sharp pain, particularly in those areas where the scalp was bruised or cut

Some tissues of the head are sensitive and others insensitive. Those which are sensitive are (i) the scalp, (ii) the periosteum, (iii) the meningeal vessels, (iv) the dura in the region of the large venous sinuses and (v) the large vessels at the base of the brain. Those which are insensitive are (i) the bone and diploe as distinct from the periosteum, (ii) the dura not in the region of the meningeal vessels or large venous sinuses and (iii) the brain tissue.

(*a*) *Neuritis of the Scalp*—Creeping feelings as if insects were moving beneath the skin and other such peculiar sensations or paræsthesiæ are probably the result of contusions or incomplete divisions of the nerves supplying the scalp. In my opinion, scalp pains resulting from traumatic neuritis are commonly the cause of so-called headaches; this diagnosis may be confirmed by injecting a ring of local anæsthetic around the base of the scalp. When testing for relief of pain by anæsthesia a generous amount of anæsthetic must be used and wide areas infiltrated, because there is a considerable overlap from neighbouring nerves in every region supplied by a specific nerve trunk. This etiology of scalp pains is similar to that of causalgia in the limbs, and particularly is this so when the skin has been lacerated and infection has supervened. Unfortunately pathological changes commonly extend along the sheath of the nerves, which do not come to rest until the optic thalamus is reached, when the condition is beyond the redemption of medical or surgical measures. When the scalp becomes attached to the bone, as it often does after suppuration or losses of tissue, there is sometimes a continuous feeling of pulling or discomfort.

(*b*) *Subdural Adhesions.*—Severe and continuous pains are occasionally due to distortions maintained by adhesions causing traction on the sensitive tributary veins draining into the large venous sinuses. Penfield and Norcross [1] were the first to describe this mechanism of pain which is thought to be brought about in the following way. During the moment of violence, whether the skull is distorted or not, the brain slides across the face of the dura at the arachno-dural interface, and for some physical reason, such as hæmorrhage, does not immediately regain its normal

[1] Penfield, W , and Norcross, N C "Subdural Traction and Post traumatic Headache Study of Pathology and Therapeusis" *Arch Neur and Psych*, 1936, 36, 75

position after the violence has ceased to act. In this position of subluxation adhesions form between the outer and inner surfaces of the arachnoid and dura respectively, and later they are stretched as the brain tends to take up its normal position in relation to the skull. Where an adhesion is attached to a sensitive area of the dura—that is, to a large meningeal artery or edge of a dural sinus—pain will be the result.

(c) *Tentorial Traction.*—Pressure or traction on large sensory nerves in the region of the tentorium produces the type of pain that is felt over the forehead or behind the eyes, this projection being explained by the fact that branches of the trigeminal nerve which supply the dura sweep backwards and then upwards and forwards over the vault to end in the frontal region. That this type of pain does occur is well demonstrated when performing ventriculography under local anæsthesia by the posterior route. After holes have been bored in the skull and an opening is being made in the dura, the patient will often complain of severe pain at the back of the eyes.

(d) *Hydrocephalus.*—Acute bursting headaches are due to attacks of acute hydrocephalus. These, however, are uncommon, as spinal or ventricular manometry rarely shows increased intracranial tension. Subnormal cerebrospinal fluid pressures, on the other hand, are much more common and are a frequent cause of headache, particularly when a fracture allows constant leakage of fluid externally or into the subgaleal space.

(e) *Subdural Hæmatomata.*—Chronic subdural hæmatomata give rise to very severe headaches, the pain being usually associated with drowsiness or with a slow pulse. In all cases of severe headaches the possibility of a chronic subdural hæmatoma should be considered.

(*f*) *Migraine and its Variants.*—Typically, a migrainous headache starts slowly, increases in severity until what may be called a plateau of intensity is reached, and then slowly recedes. One or, as is common, both sides of the head may be affected and the pain may radiate into the neck, ears or face. Visual hallucinations such as black spots or coloured lights, may or may not accompany the attack. Often when the pain is at its height the patient is completely incapacitated, feels confused and has to lie down or go to bed to get relief; vomiting or nausea often occurs. The headaches tend to have a definite periodicity, coming on in the morning, afternoon or evening; they are brought on for no particular reason. Such headaches occasionally occur as a sequel to acute cerebral trauma. Again, typical migrainous headaches may be aggravated by the injury, inasmuch as they occur more frequently and last for a longer time.

We come now to the crux of the problem and to consideration of cases in which the headache does not fit into a formal clinical or pathological group. In order to obtain a broad perspective of the subject let us review the development of the headache syndrome from the acute phases of injury.

We know that a blow on the head is painful; for example, a hæmorrhage into the temporal muscle which stretches the temporal fascia causes severe aching pains until the hæmatoma resolves and the fascial stretch is released Also, a violent shake of the head can lead to headache without bruising of the soft parts.

In severe head injuries when the patient has been rendered unconscious, his movements and facial expressions often suggest that he is in pain. Subarachnoid hæmorrhage in cerebral trauma is common, and we know that in rupture of congenital aneurysms of the circle of Willis severe head pain is an outstanding feature of the illness. There is indeed abundant evidence to prove that the acute stages of head injury are painful. It might, therefore, with reason be suggested that post-concussional headaches are due to continuing action of those factors which cause head pain in the subconscious phases.

Immediately after the recovery of consciousness a patient makes a specific complaint of head pain only when closely questioned. Without leading questioning he usually volunteers the information that his head "feels funny," that he feels ill and cannot think properly. Later he complains of headache. The ache is usually of a general character although there is often a particular stinging over the region of a bruise of the scalp or a fracture of the vault. The headache lasts a few days or a week and then begins to recede and may completely disappear at the end of one, two or three weeks. In my experience it is rare for a patient at this early stage to have a blinding type of headache which makes him curl up in bed and ask for medicine to give him relief. When this does happen, the patient has either had a severe subarachnoid hæmorrhage or normally has a tendency to severe migraine, and such an attack has been precipitated.

Headache in the convalescent phase is due to bruising of the soft tissues, or to subarachnoid bleeding or to precipitation of a constitutional tendency to headache.

At the end of the convalescent period the patient is sent either to his home or to a rehabilitation centre. Unfortunately little is known of the patient who returns home until he appears later for review. During the early periods of skilled rehabilitation a patient does not as a rule complain of severe headache. He does, however, commonly state that his head does not feel the same. He is "conscious of it" and at times it "feels fuzzy."

Let us now go a further step forward and consider the cases that come for review after a period of rest or of formal rehabilitation, say two months after receipt of injury The patients may or may not have been seen in the acute stages of injury It is of great help in the assessment of post concussional symptoms if the patient has been seen in the acute stages For example, a head injury might have been so trivial that it could not possibly have caused structural changes sufficient to cause persistent pain. On the other hand, lumbar puncture and cell count in the acute stages may have proved the existence of a severe subarachnoid hæmorrhage which caused severe headaches similar in character and distribution to those of which the patient now complains

No sex nor age group is immune from post-concussional headaches Headache in young children after head trauma is common, and mothers will testify that their children often come in from play asking to lie down, which is a commentary on the genuineness and severity of the head pain The child indicates the site of the pain with a sweep of the hand, usually across the forehead, denoting that the pain is frontal Relief comes with sleep The pain is essentially periodic, the child being quite well in the intervals save perhaps for a tendency to irritability

In old people, headaches are apt to be continuous and associated with noises in the head Possibly all the injury does in many of these cases is to call attention to pre-existing changes resulting from cerebrovascular degeneration

It is however, the young adults and the middle aged who form the largest group presenting the headache problem, not only because of their numbers but equally for economic considerations These cases fall into two distinct groups.

Group 1 embraces those patients who remain incapacitated and have suffered from headache since the recovery of consciousness

The head pain may or may not be similar in character to that which was felt in the convalescent stages of injury In practice the characteristics of the pain are rarely volunteered by the patient and descriptions have to be sought by the examiner through leading questions. Even then only a very vague impression of what the patient is suffering can be gained The pain may be a deep ache or a pressing or a bursting sensation Complaints of sharp pains alone are rarely heard Usually there is a background of generalised discomfort associated with one or more particularly painful areas from which pains occasionally shoot to other parts of the head Tender or hyperæsthetic areas are commonly found on palpation of the scalp Headache in this first group is rare as an isolated symptom Usually the well known signs and symptoms of altered disposition nervousness

and lack of concentration, etc., are present, the headache being merely one feature of a confused symptom complex. To make the problem more difficult, many of the patients are awaiting some kind of compensation, from the Workmen's Compensation Act, or Civil Law or by private arrangement.

To attempt to cure a headache only in this type of case is profitless. The whole symptom complex must be treated if satisfactory results are to be obtained. As a guide to therapeusis, certain principles may be laid down :—

(i) The doctor must give his patient confidence in his own medical skill This necessitates an understanding of the illness and a desire to get the patient better.
(ii) Litigation worries should be early and satisfactorily settled.
(iii) Frank organic lesions such as chronic subdural hæmatomata or hygromata must be eliminated and if necessary drained. Such lesions are rare.
(iv) The patient should be removed from the environment which tends to perpetuate his symptoms.
(v) Correct rehabilitation is essential.

These aims are, of course, ambitious, and under present conditions difficult to attain.

Functional and organic factors so interplay that any kind of anxiety state is apt to perpetuate or aggravate a simple headache mechanism. Headaches are commonly made worse by domestic worries, and in Service cases by a desire to escape danger or other uncongenial circumstances.

Group 2 embraces those patients who, after accident, have made a reasonable recovery and have returned to some form of useful work or training, later to break down or to complain persistently of headaches. It will be assumed that psychological factors which precipitate or perpetuate head pains have as far as possible been minimised by

(*a*) Correct medical and surgical procedure in the first instance
(*b*) Correct treatment in the convalescent stage.
(*c*) Early settlement of the man's worries and reassurance.
(*d*) Correct rehabilitation

Headaches of all types, grades of severity and duration are described. This is what might be expected, for even though the pain mechanisms were of the same type and severity in all cases. the reception must differ as do personality and intellect. In other words, there are two variables—the nature of the pain and the mental build of the patient. None the less certain useful generalisations regarding the pain can be laid down.

Mode of Onset—The pains come on more often suddenly than slowly, which is of course unlike a typical migraine On the other hand, in many cases when they have once started they tend to get worse

Time Incidence and Precipitating Cause—They may come on at any time of the day Usually they are precipitated either by physical exertion, noise or mental concentration of the simplest type such as reading

Relief—This is best obtained by sitting quietly or going to bed Few patients resort to analgesics, such as aspirin

Characteristics of the Pain—The characteristics of the pain are usually very vaguely described by the patient. The discomfort is essentially an ache and any part of the face may be affected There may or may not be sharp stabs. Specific terms such as boring, pressing etc., will on close inquiry often be found to be the words of the examiner, not of the patient Description of a headache is, in fact, a most difficult exercise even for one trained in medical observation. Vagueness is no measure of genuineness Pain may radiate from one position to any other part of the head, into the ears, eyes or neck.

Duration and Frequency—Most patients appear extremely puzzled and are unable to give a clear-cut answer to the simplest question of how frequently their pain occurs and how long each painful episode lasts If patients' statements on this point were accepted at their face value then it would have to be assumed that many of them suffer no more than a few moments' pain once or twice a week. In fact, the pain comes on whenever conditions for that type of person are adverse and only disappear when favourable conditions are regained This important information usually has to be dragged out of the patient.

Associated Signs—Associated signs such as vomiting changes in colour or heaviness about the eyes are rarely seen during an attack of headache When these do occur they are reliable indications of the genuineness and severity of the pain

The effect of the pain is variable In few cases, however, is it sufficiently severe to cause a patient to go to bed In industry the afflicted usually continue with their work save for occasional days off although sometimes the pains are so incapacitating that less noisy or lighter forms of employment have to be sought Women carry out their housework regularly but with little zest or pleasure

In my experience few Service cases refuse their passes out of hospital to go into the noisy traffic of a town On the other hand they commonly return after being to a cinema complaining

of headache and lie down on their beds. The complaint of these men is that they are unable to carry out strenuous duties efficiently.

In summing up it can be said that in all grades of life the type of headache now under review interferes with positive health and only occasionally is frankly incapacitating.

The underlying causative pathology is extremely difficult to determine. In the typical case there are no abnormal neurological signs. The cerebrospinal fluid pressure, as measured by manometry, lies within normal limits Encephalography proves that there is no space-occupying lesion within the skull, such as a subdural hæmatoma. The cardiovascular and other systems are normal. As far as can be judged, the pains are rarely due to (i) neuritis of the scalp, (ii) subdural hæmatomata, hygromata or adhesions or (iii) hydrocephalus.

As we are dealing with purely subjective phenomena, there is always a suspicion that a man might be a malingerer A Service case may attempt to escape danger and place himself in more congenial surroundings The belief that malingering is rare is probably correct, but it must be realised that it is founded on no sound scientific basis. Sometimes the pains are purely psychogenic in nature; that is, they arise in the mind and not in the body of the patient. Complaints of headaches are a common way of expressing a feeling of mental ill-health.

Finally, let us consider the mechanism of the pain in migraine as this may throw some light on the present problem. There is much evidence to suggest that the pain in migraine is due either to extreme spasm or distension of the arteries of the scalp, of the dura and of the circle of Willis, also, the trigeminal pathways are those by which the pain is usually conducted to the brain. The essential disturbance which causes the blood vessels of the head to go into spasm or distension is not known. Here a certain amount of conjecture is necessary but, I think, justifiable. The hypothalamus is the highest controlling centre of the vasomotor system. From the hypothalamus messages are transmitted through the brain stem and spinal cord by pathways as yet uncharted to the peripheral autonomic nervous system. The sympathetic nerve supply in the blood vessels concerned is shown in Fig. 233. It is my belief that in many cases the equilibrium of the hypothalamus is rendered unstable by injury and on occasions reacts in an abnormal way to the many stimuli, emotional or chemophysical, which affect it. A dysrhythmia is aroused or, in other words, an abnormal message is transmitted which leads to painful spasm of the blood vessels of the head and to the peculiar feelings which accompany them.

Post-concussional head pains, therefore, I believe in many cases have a physical basis, even when the essential precipitating factor is an anxiety state or the desire to escape danger or uncongenial duties

2 **Dizziness** [1,2]—Dizziness, strictly speaking is a hallucination of movement in oneself, in one's environment or in the third dimension which interferes with or tends to interfere with the sense of balance This type of disorder is relatively rare and is due to a hæmorrhage into the labyrinth or to damage of the labyrinthine fibres of the auditory nerve The symptoms are similar to the crises of Ménière's disease in which the patient is seized with an uncontrollable giddiness and falls heavily to the ground without losing consciousness In the less severe attacks the ground seems to lift a little, causing a sense of insecurity

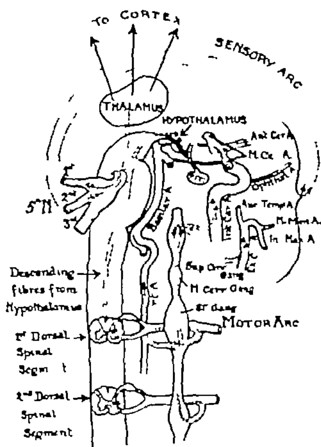


Fig. 233

The neural mechanism of migrainous pains

Abnormal messages arise in the hypothalamus and are transmitted by the central and peripheral pathways delineated above to the large vessels of the head. This is the motor arm of the reflex arc of migraine The painful stimuli aroused by excessive spasm and distension of the arteries concerned are chiefly conveyed to the brain by the trigeminal pathways This is the main sensory arm of the reflex

Physical or neurological evidence of labyrinthine dysfunction in between attacks is often meagre and in fact may not be demonstrable by simple clinical means particularly as associated cochlear involvement rarely causes gross impairment of hearing Before making a final pronouncement on the origin of dizziness, detailed radiographic studies of the petrous bone will have to be made and the integrity of the semicircular canals and cochlea determined by caloric tests and audiometer readings respectively Fractures of the petrous bone are often incomplete, the

[1] Cawthorne T and Cobkey F S "Vestibular Injuries" *Proc Roy Soc* 1946, 39 p 270
Phillips, D G "Investigation of Vestibular Function after Head Injury" *Jour Neurol, Neurosurg and Psych* 1945, 8, Nos 3 and 4.

of headache and lie down on their beds The complaint of these men is that they are unable to carry out strenuous duties efficiently.

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corneal reflex on the affected side papilloedema and increase of proteins in the cerebrospinal fluid Thrombosis of the posterior inferior cerebellar artery is often heralded by the most violent attack of giddiness, but the diagnosis is usually a simple matter, since this condition not only produces difficulty in swallowing but also sensory changes on the whole of one side of the body or on the face of the same side and in the limbs and trunk of the opposite side Linthicum and Rand[1] are convinced that labyrinthine changes after severe concussion are commonly demonstrable and much has been written on this subject by the French school of neurologists[2-4]

Usually what the patient means by giddiness is that things go black before his eyes whenever he makes a sudden movement, such as stooping From the table on page 353 it will be seen that this sensation is far more common than actual hallucination of movement. Upward movements of the eyes are particularly liable to precipitate an attack These momentary black-outs are probably due to instability of the cerebral circulation, consequent upon injury to its vasomotor apparatus rather than to labyrinthine dysfunction Thus the essential cause of most giddy attacks is momentary ischaemia of the brain consequent on circulatory lability

Ataxia and loss of balance due to gross lesions of the cerebellum or its connecting pathways are exceedingly rare In any case these dysfunctions are chiefly objective rather than subjective in nature From the prognostic point of view accurate diagnosis is essential since circulatory disturbances usually recover spontaneously, whereas physical injuries to the labyrinth often necessitate surgical intervention, such as division of the labyrinthine fibres of the eighth nerve or complete destruction of the labyrinth with alcohol before relief can be obtained

3 **Insomnia.**—Insomnia is a most troublesome symptom not only in itself but inasmuch as it gives rise to a vicious circle So long as it exists recovery of general health is improbable yet on the other hand insomnia is unlikely to improve so long as the patient feels mentally and physically ill Whether insomnia is the result of structural changes within the brain or to anxiety combined with a general deterioration in health is not known. Probably it is due purely to psychical processes interfering with the normal physiological integrations on which sleep depends Usually a patient cannot get off to sleep till the early hours of

[1] Linthicum, F H., and Rand, C. W "Neuro-otological Observations in Concussion of the Brain." *Arch. Otolaryng.*, 1931 13, 783.
[2] Barre J A. *Rev d'Oto-neuro-opht.*, 1932, 10, 258.
[3] Lortman, O., and Delmas-Marsalet, P *Rev d'Oto-neuro-opht.*, 1933, 11, 23.
[4] Ramadier J., and Caussé R. "Traumatismes de L'oreille *Masson et Cie* Paris 1937

the morning, and when he does he soon wakens again to start the same process once more, with the result that he wakens in the morning feeling more fatigued than when he went to bed. Often he will get up in the night and make himself a cup of tea. Nightmares in which the details of the accident are reproduced are infrequent, although disturbing dreams in which something sinister happens are fairly common. No doubt the rarity of reproduction or imaginary repetition of the details of accident is linked up with the state of retrograde amnesia, that is, blankness of memory for events immediately preceding unconsciousness. In people over sixty, especially those with high blood pressures, even minor injuries may result in intractable insomnia

4. Diplopia.—In the acute stages of severe concussion, when a patient is still unconscious, skew deviation and inco-ordinate wandering of the eyes are often seen. Probably they are due to disturbances of the ocular nuclei within the brain stem, caused by petechial hæmorrhages or faulty circulation in the radicles of the basilar and cerebellar arteries. Most cases of double vision are the result of the continuation of this state, although in a lesser degree. The fault in the affected muscle is often so slight that it is difficult to prove which muscle is causing the diplopia Recovery in this type of case is spontaneous and rapid.

Gross paralysis of extraocular muscles, causing an easily discernible squint, is usually due to injury to the trunks of the third, fourth or sixth nerves themselves as they enter or traverse the orbit. Rotations of the brain stem occasionally lead to attrition of the sixth nerves as they lie within the posterior fossa. Recovery in these cases is doubtful The prognosis is favourable when improvement starts early or when the paralysis is partial, but it is doubtful if no improvement has occurred within three months.

Fractures frequently involve the orbit, causing deformity of the orbital cavity with resulting displacement of the eye. Such a deformity, however, is not necessarily accompanied by diplopia, because the brain is often able to compensate for the displaced eye by fusing the images which come from it with those from the other eye, or by suppressing them altogether. Compensation, however, rarely occurs when neuromuscular mechanisms are permanently affected.

5. Changes in Disposition.—The anatomical and physiological basis of emotion is thought by some to be seated in the thalamus and other centrally placed nuclei Outbursts of rage occur in patients with tumours affecting the hypothalamus, and surges of uncontrollable temper and homici[illegible]ndencies have been observed in cases of disease of th[illegible]us. Similar states

occasionally follow head injuries possibly due to small hæmorrhages in the thalamus and hypothalamus.

The usual clinical picture, however, is not one of violent extremes. It is that of a previously good tempered happily minded man becoming irritable, intolerant, selfish, disinterested and miserable. Instead of being concerned with the well being of his family he will refuse to work and by his behaviour will focus the whole attention of his wife, relations and friends on himself. He loses interest in the things which used to engage his attention and will sit for hours gazing into the fire without troubling to answer when spoken to. He finds noise of any kind irksome, he is peevish and avoids social contacts. Self confidence and self reliance completely disappear. There are, of course, changes of a lesser degree which are obvious to intimate relatives only.

Of all the symptoms of the post-concussional syndrome changes in disposition are the most distressing to the family concerned, particularly as complete recovery is often not made. The underlying pathology of this condition is probably the result of widespread physical damage in the brain of a diffuse contusional nature.

6 **Intellectual Changes**.—The link between the brain and the mind is not known but since intellectual processes are dependent to some extent on the experiences of the special senses which are represented widely over the brain it is reasonable to presume that the whole brain rather than parts of it are concerned with the intellect. Possibly the frontal lobes are concerned only with the highest intellectual activities.

Gross impairment of intelligence is uncommon in the post concussional state. When it does happen it is due to laceration of the brain or in other words to gross loss of cerebral tissue.

The usual picture is that of mental sluggishness—an unwillingness to think rather than a total inability to do so. A patient will answer questions slowly but correctly even if this is done in a roundabout way and he will reason and converse in a perfectly logical manner. In fact his theorising on the cause of his symptoms is often illuminating. He is accessible and can always be made to understand though not necessarily convinced. Improvement rapidly takes place.

In children of school age intellectual changes are common and most serious[1]. At the very least the child's educational training is retarded by the length of the illness which is often the equivalent of one year for even though the child returns to school in a

[1] Blau, A. Mental Changes following Head Trauma in Children. Arch. Neur. and Psych., 1936, 35, 723.
Paterson, A. and Zangwill, O. L. Recovery of Spatial Orientation in the Post Traumatic Confusional State. Brain, 1944, 67, p. 54.
Tooth, G. On the Use of Mental Tests for the Measurement of Disability after Head Injury. Jour. Neurol., Neurosurg. and Psych., 194[illegible], 10, [illegible]

few months his new studies are incomprehensible to him without the earlier grounding leading up to them. The child gets behind, is thrown with strange companions and becomes discouraged In any case, if he is not kept at school beyond the usual leaving age for a period equivalent to that which he has lost, his final educational standard must be lower than it would otherwise have been. The parents of children of the upper and middle classes are well aware of this fact and act accordingly, but unfortunately the children of the working classes are usually not safeguarded in this respect as they should be.

Before school age the effects of injuries on the intelligence cannot be assessed save in those cases where they cause frank mental deficiency.

7. **Neurosis.**[1]—By definition a neurosis is a disorder of the personality not based on physical injury within the brain of any particular pattern. On the other hand it is often the result of the reaction of the mind to objective or subjective effects of a structural pathological state in the brain. Every neurosis, however, following an injury to the head does not necessarily imply a physical injury to the brain, as the state may be produced purely by psychological trauma or the mental shock of the accident. Prolonged trauma, such as occurs in industrial accidents when a man is trapped and crushed and does not lose consciousness immediately, is almost invariably followed by a functional overlay with the attendant train of symptoms of which loss of confidence and tremor are the predominant features.

Purely neurotic syndromes following minor injuries are common, and the prognosis in these cases is capricious, since the breakdown may be due mainly to business worries, domestic unhappiness or frustration of ambition, and these conditions must be corrected before improvement can be expected.

The underlying cause of a neurosis syndrome is a matter for speculation. The symptoms, of course, result from excessive reaction to physical, mental, social and economic influences. In some cases careful inquiry will reveal an obvious predisposition to the illness. One or both parents may be unstable; this weakness is transmitted at birth to the child in whom the tendency is fostered by witnessing the general behaviour of the parents and their reaction to trying circumstances. In many cases of post-traumatic neurosis a frank anxiety complex is active That is, there is an exaggerated and unduly prolonged reaction to a sense of fear which comes with the realisation of what has happened to them. This leads to a persistent sense of uncertainty and loss

[1] Schilder, P "Neuroses following Head and Brain Injuries" "Brook's Injuries of the Skull, Brain and Spinal Cord" Baillière, Tindall & Cox London 1940

of confidence. For some time the patient is able to disguise his uneasy feelings but sooner or later the illness makes itself felt, not only in the mind but on the body as well. Exteriorisation then takes place, the illness being expressed in a way other people can understand. The patient loses his power of concentration and cannot sleep; his movements become tremulous and he develops headaches, giddiness and other disabilities. Later he forgets that it was his anxiety or sense of fear that caused his lack of concentration and headache and begins to think that his illness does not consist of but is due to these symptoms. By this means he is able to convince himself that he is not responsible for his behaviour and cannot reasonably be asked to do even the lightest form of work.

Possibly the essential psychological mechanism in an anxiety neurosis is mental conflict[1], a battle of the primitive against intellectual influences. In other terms it is an unsettled argument between the urge of self preservation plus the desire to live a smooth, congenial, stable life on the one hand and a sense of duty, pride and citizenship whatever the cost on the other.

Breakdown of personality can also be caused by persistent physical ill health and the stress of adverse social and economic conditions. Possibly the mechanisms are similar to those acting in anxiety, the ultimate object being escapism. A strong argument against this suggestion is that personality breakdown is extremely rare in severe chronic physical afflictions not resulting from injury. Also adverse circumstances such as lack of education and poverty in many people act as a stimulus to do better.

Again, to argue from the other side, most clinicians believe that the brain is concerned with thinking, on which personality is essentially based. If this is true then it is possible that personality can be directly influenced by a physical injury since this can alter the state of the brain.

Finally we have to consider the importance of the desire for compensation following injury. Experience has proved beyond all doubt that it can aggravate and perpetuate almost any kind of psychoneurotic state. The repayment for recovery might have to be made in pounds, shillings and pence in sympathy or in retribution. Whether a desire for compensation can actually cause a psychoneurosis—the new and more correct title of neurosis—is not known; probably it can and commonly does. However until the methods of arbitration and modes of living have been substantially altered it will be quite unjustifiable to classify

[1] Symonds, C. P., "Neurosis in Flying Personnel," *Brit. Med. Jour.*, 4th Dec. 1913, p. 03; "The Human Response to Flying Stress," *Brit. Med. Jour.*, 11th Dec. 1913, p. 740; "Anxiety Neurosis in Combatants," *Lancet*, 1913, 2, [illegible]

many cases of "traumatic neurosis" as a form of malingering. What is certain is that psychoneurosis colours almost every post-concussional picture.

This subject of neurosis and psychoneurosis has become so important that a new chapter is devoted to this problem.

8. **Psychoses.**[1-4]—These may be primary or secondary. In the first case the mental disorder is due directly to a physical injury of the brain, whereas in the second case a pre-existent degenerative process or latent psychotic state is merely precipitated by the injury.

Primary.—Typically, the sequence of events in a primary psychosis is as follows. A patient is rendered unconscious by a blow on the head. Instead of approaching consciousness through the ordinary phases of irritability he becomes raving mad and so uncontrollably dangerous to himself and those around him that he has to be transferred to a mental institution where better facilities obtain for the treatment of this kind of patient. As the man becomes more conscious it will be seen that he is grossly disoriented regarding time and place and is quite oblivious of his circumstances, although he may evidence distress regarding physical discomforts. Usually with time and rather rapidly he regains insight, becomes biddable and very soon is normal as far as his psychotic state is concerned. Only occasionally does the mental state retrogress, the man becoming an obvious lunatic. In this state his physical condition also deteriorates. He becomes incontinent, his skin sags, he loses weight; also his circulation becomes sluggish, as shown by cyanosed extremities and a blotchy complexion. The prognosis of a primary psychosis in the absence of a history of mental instability is good.

Secondary.—Secondary psychosis may be subdivided into two groups. The first group embraces those cases which fit into a formal classification of dementia præcox, paranoia and mania, etc. They are merely precipitated by the injury, as shown by the fact that they may occur after trivial injuries to the head which could not have produced cerebral damage sufficient in degree or extent to account for them.

In the second group a degeneration of the nervous elements takes place after an interval of a few weeks or even years, and this proceeds far beyond the limits of the original injury, leading to profound mental defects, which declare themselves clinically in a

1 Symonds, C P "Mental Disorder following Head Injury" *Proc Roy Soc Med*, 1937, 30, 1081

2 Goldstein, K "After-effects of Brain Injuries in War" *Wm Heinemann* London, 1942

3 Walshe, F M R "Diseases of the Nervous System" E & S Livingstone Ltd Edinburgh, 1941 Second Edition

4 Curran, D, and Guttman, E "Psychological Medicine" E & S Livingstone Ltd Edinburgh, 1943

variety of ways (Fig. 234). A good example of this secondary change is a condition known as "punch drunk," seen at the end of a long career in boxing. Repeated jolts to the head cause petechial hæmorrhages into the brain substance which at the time pass unnoticed but which may later lead to mental impairment and loss of vision.



Fig. 234

A ventriculogram of an advanced case of diffuse cortical atrophy following trauma. The ventricles are many times larger than normal. View: anteroposterior shoot. The outlines of a normal-sized ventricle are sketched in.

In arteriosclerotics, syphilitics and alcoholics, trauma to the head is particularly liable to initiate a diffuse and degenerative process in the brain tissue. Minor degrees of violence that would cause no serious structural damage in a healthy brain may, under the foregoing conditions, lead to *progressive insanity*. In the primary and formal psychotic groups accompanying neurological signs of damage to specific parts of the brain are sparse or absent, whereas they are marked in the secondary degenerative type of case. Prognosis in a primary traumatic psychosis is good, poor in a formal psychosis and hopeless when a degenerative encephalopathy is present.

THE EXAMINATION

Let us start at the moment when a man, complaining of the results of an injury to his head and with litigation at stake, walks into the consulting room. Usually he enters slowly and hesitantly, accompanied by his wife and often with other relatives or friends He will stand about aimlessly until told what to do, and will rarely attempt to take off his overcoat or get rid of his hat until his wife does it for him; she also sees him safely to his seat. Characteristically, he will sit perfectly still staring straight in front of him with a fixed expression of peculiar indifference on his face It is the demeanour of his wife that shows how profoundly unhappy they both are. When invited to tell his story he rarely speaks spontaneously and says all he wants to say in a very few words. When asked a question he will almost invariably wander round the point and even when pressed rarely gives a straightforward answer Incessantly he will appeal to his wife to answer his questions for him; many times she will do this without invitation or against instructions not to do so. Rarely from the man alone is it possible to get reliable details of his ailments or of his convalescence. Often he will omit to mention one or more of his more prominent symptoms; also, symptoms are described in the vaguest language. His attitude to the doctor appears to be one of: "You don't understand nor does anyone else, and I can't tell you; and if I could it wouldn't do any good." His wife, though rarely voluble, gives her opinion in a very earnest manner, being obviously gravely concerned about the whole affair. Finally the man usually complains of pains in his head, giddiness and insomnia or occasionally of something specific, such as deafness and loss of taste It is, however, from the wife that the true state of affairs is ascertained. From her it will be understood that her husband is a changed man both mentally and physically. He has lost his initiative and his interest in everything and everybody. He will sit for long periods staring in front of him without saying anything to anybody. He is unfriendly and avoids social contacts He is irritable, loathes noise, objects to the wireless and is peevish with the children. From his wife he expects and demands the minutest attention. In fact he becomes a profound valetudinarian. He is unable to concentrate; for example, he will read a few words of the paper and then put it away. Memory for recent and immediate events is faulty. Typically he will go into the next room to get something and return without it, having forgotten what he went for and apparently quite oblivious of the fact that he did go for something. If sent on a message, instructions have

to be written down for he would not remember what they were He sleeps badly, for long periods he remains awake and often gets up to make a cup of tea for himself Later he returns to bed and finally does not get up until noon or thereabouts to start his life of lack of purpose once again. Usually his appetite is not impaired His general health looks excellent and he is often tanned in comparison with his pale-faced wife and fellow workmen Sex relationships radically change, a passionate attentive man will become indifferent to his wife and often brutal in his behaviour

Volubility on the part of the patient when taking a history is rare, but emotional breakdowns, tears and despairing gestures are common

After the taking of the history there starts the fuss of getting ready for the examination Speed of undressing in my experience is a social distinction A working man always does this slowly and often loses his stud, whereas a professional man undresses rapidly When complaining of the post-concussional syndrome any patient takes an unconscionably long time to take off his clothes and has to be helped by his wife As a matter of interest I once allowed a man to undress and dress at his own speed and timed him He took exactly twenty minutes to undress and half an hour to dress If spoken to when about to untie his bootlace a patient will stop all movements sit upright, think about what he is going to say and then say it slowly When undressed he will complain of the cold and his wife will give you the impression that you are doing him serious harm by stripping him It will be found that his gait is slow deliberate and a little unsteady when on the turn. Bending and rotation tests are carried out slowly, clumsily and gingerly It is, however rare to precipitate a true attack of vertigo by movement and although a patient may stumble a little he rarely falls or lurches badly Looking up may cause him to bend his head and put his hands over his eyes Nystagmus of an abnormal type is rarely seen Usually co-ordination of movement is grotesque There is often a gross ataxia when attempting to touch the nose with the finger or the knee with the opposite heel also, the patient may tremble all over his body when asked to do things a little more expeditiously

Occasionally a patient will roll and lurch about the room knocking everything down but himself or may shiver so violently that things in the room vibrate On attempting to manipulate the legs they are held voluntarily rigid The knee jerks may be hyperactive or obliterated by voluntary spasm The plantar responses are almost invariably flexor even when the knee jerks are wildly exaggerated Ankle clonus is usually absent But what is interesting is that the abdominal jerks are often absent or

inactive. Frank paralysis of the limbs or areas of true anæsthesia about the body are exceedingly rare. The heart rate is fast but varies according to the stage of the examination at which it is taken. At first it may be racing, but later, as the patient's confidence is gained, it often settles down to normal, save in those cases where a general tremor persists. Except in conditions of frank poverty a man rarely loses weight because of his illness. When he does he looks very ill. Menstruation in young women often becomes irregular or disappears for a time. In the several pregnant women who have been under my care, accident has not brought on parturition. The optic discs rarely show any change. The fields of vision are reputed to be constricted, but apart from patterned defects I have been able to place little reliance on charting of the visual fields because of the patient's stupidity, unwillingness or inability to co-operate Blood pressure in the absence of circulatory disease is rarely raised.

The above description is, of course, of a prototype embracing the worst features of the post-concussional syndrome At the other end of the scale is the soldier who, without exaggeration of word or deed, declares that he has headaches or suffers from dizziness or other such symptoms.

Apart from destruction of localised parts of the brain or special organs producing such obvious conditions as hemiplegia, aphasia or deafness, the summary of the neurological evidence in the majority of cases is mental and physical sluggishness and dispositional changes. It will be realised, therefore, that in the absence of localising signs further opportunities for observation and investigation will be necessary before a final opinion can be made on the genuineness of the symptoms and what the nature of the underlying pathological change in the brain might be.

SPECIAL INVESTIGATIONS

Radiography.—Though a fractured skull in itself is of no very great importance, it does point to the fact that the head has been subjected to a considerable amount of trauma. Moreover, radiography may reveal injuries to specialised structures such as the middle and internal ear or the optic canals which not only confirm that certain symptoms are genuine but also explain the mechanism by which they were brought about.

Lumbar Puncture and Manometry.—At the time of lumbar puncture the pressure of the cerebrospinal fluid should be measured so that symptoms such as headaches, the result of increased or decreased intracranial tension, may be eliminated or confirmed. In sixty patients suffering from the post-concussional syndrome

the results of spinal manometry were as follows in one the pressure was high, in thirty the pressure was normal, and in twenty nine the pressure was in the low normal register normal pressure in the lateral position being regarded as lying between 50 and 150 mm. of cerebrospinal fluid Low pressures were most commonly found after cerebrospinal fluid leaks and in debilitated patients of poor nutrition and weak circulations High pressures are only found in patients with frank pathological changes in their brains, such as hydrocephalus and subdural hæmatomata or hygromata In all cases, cytological and chemical examination should be made of the cerebrospinal fluid, since abnormal findings may lead to the discovery of an unsuspected lesion such as a neoplasm.

The Queckenstedt Test.—Bilateral compression of the jugular veins causes cerebral congestion and a rise in intracranial pressure This pressure is transmitted to the spinal theca and can be measured by spinal manometry Normally on bilateral compression of the jugular veins the column of fluid in the manometer will rise at least 20 cm in ten seconds and fall to the original level within fifteen seconds. Any deviation from normal in the way of a diminished rise, of a slow or of a stepped fall, is indicative of obstruction within the cerebrospinal fluid pathways, this usually is of the nature of meningeal adhesions The importance of demonstrating the presence of adhesions is that they may be the cause of meningeal pain or may produce attacks of so-called acute or transient hydrocephalus with headaches of the bursting type

Encephalography—When localising neurological signs are present or intractable pain or other persistent symptoms are complained of an encephalographic examination should always be carried out, otherwise gross pathological lesions not detectable by clinical methods may pass unnoticed In particular thin surface clots or subdural collections of fluids may be demonstrated the drainage of which will lead to cessation of symptoms Also, a man s general intellectual and locomotor poverty may be shown to be attributable to a diffuse loss or atrophy of cerebral tissue as shown by enlargement of the ventricles, basal cisterns and gyral spaces

Electro-encephalography—Electro-encephalography will soon become a routine method in the investigation of post-concussional phenomena. The following conclusions of Williams[1] summarise the current opinion on the subject in this country —

1 After a head injury an abnormal electro-encephalogram usually indicates organic cerebral abnormality resulting

[1] Williams D "The Electro-encephalogram in Chronic Post traumatic States" *J Neur and Psych* 1941 4, 131

directly from the injury. This abnormality is present in half the cases of the chronic post-traumatic syndrome

2. Clinical recovery and improvement in the electro-encephalogram are usually closely related, and so long as the relationship holds, the prognosis is good.
3. Abnormality may persist in the electro-encephalography in any one patient after clinical recovery appears complete. This indicates residual cerebral dysfunction which is not demonstrable by the usual methods of psychological investigation. It is usually followed by complete recovery, but the abnormality may explain the relapse of some patients after they have returned to their employment.
4. A normal electro-encephalogram after a head injury almost invariably indicates absence of abnormal cerebral tissue, which may be due either to full resolution or to complete destruction of damaged cerebral substance. When a normal electro-encephalogram is associated with full clinical recovery prognosis is consequently good, but when accompanied by persistent symptoms the ultimate prognosis may be bad.
5. There does not appear to be any difference between the electro-encephalogram of traumatic and idiopathic epileptics, and prediction of traumatic epilepsy does not seem possible by electro-encephalography during the period of recovery from a head injury.
6. The electro-encephalogram appears to reflect the degree of cerebral dysfunction resulting from trauma with considerable accuracy, and it has a useful place in the management of cases of head injury when used in conjunction with other methods of clinical examination.

General Metabolic Overhaul.—The danger of specialism is the tendency to attribute every symptom to the nervous system, forgetting that other organs of the bodily economy may be at fault. Therefore a full examination should be made of the respiratory, circulatory and urinary systems and of the blood, including cell counts and the Wassermann test. Bright's disease, malignant circulatory hypertension, syphilitic arteriosclerosis, diabetes mellitus and other disorders may easily be overlooked if their possible presence is not sought by the usual simple methods It is well to emphasise that the urine should be examined and the blood pressure taken as a routine measure.

Psychological Overhaul.—Whenever a frank and troublesome functional element is present the help of an experienced

psychiatrist should be sought and the patient passed over early to him for treatment if it is the opinion of the doctor that medical and surgical measures associated with time and rehabilitation will not effect a cure

DIAGNOSIS AND ASSESSMENT OF SYMPTOMS

When all the clinical and investigational data have been collected the next problem to be solved is whether the symptoms complained of are genuine, and if they are, what the underlying pathological state which causes them may be

First it is important to get a clear picture of the man's pre-accident background Was there evidence of a neurotic tendency ? Was he a good husband ? Was he a good workman ? Usually this information is given faithfully by his wife or relatives con firmation easily being obtained by consulting his employer Poor types commonly have a characteristic stamp about them but it is very wrong to believe, as do so many clinicians, that it is possible to weigh up a man's character in a few minutes. Such hasty judgments often lead to grave injustices Also, it must be realised that an ill educated working man is at a great disadvantage when attempting to state his case, being apt to irritate the examiner unduly

The next point that must be determined is the nature and severity of the original illness. In particular, did the man receive an actual cerebral injury and of what degree ? In cases of closed head injuries this has to be judged by the length of time he was unconscious In compound injuries the surgeon's notes will usually supply the necessary information In my opinion an injury which leads to unconsciousness of more than an hour s duration is always associated with a diffuse neuronal injury of a structural type in which many cells are permanently destroyed A man who receives such an injury is not able to sit up immediately moreover he is more than confused when admitted to hospital and does not speak coherently for many days Unconsciousne s which develops after a latent interval is not due to intrinsic damage and therefore is not so liable to be associated with permanent structural changes within the brain With a knowledge of the type of man we are dealing with and of the nature of the injury he received we are better able to understand the effects of the injury

For example when it is known that a man complaining of aches and pains was well balanced mentally before his accident and has received an injury to his brain of the structural type then it is reasonable to presume that some of his symptoms at least have an organic basis On the other hand, functional

influences must account for symptoms following minor injuries, particularly when they occur in a man of pre-accident neurotic tendencies. An organic basis may also account for vague symptoms in those cases where there is frank evidence of local brain damage, such as hemiplegia, hemi-anæsthesia, aphasia or loss of smell

Whether headaches are due chiefly to neurotic or to organic influences is almost an insoluble problem, for the good reason that they are so essentially subjective in nature As far as I can see, when there is an element of real pain in contradistinction to an ache, the pains are usually of organic origin. Evidence of meningeal adhesions or hydrocephalus, of course, confirms this view. On the other hand, dull aching pains of an indefinite character unrelated to the time of day, climate,[1] excitement or exertion are often neurotic in origin.

Dizziness is almost always due either to instability of the cerebral circulation or to contusion of the labyrinth or eighth nerve.

Apart from frank intellectual impairments due to bilateral frontal lobe contusions or to degenerative encephalopathies, it is difficult to attribute faulty memory and slowness of the mental processes to anything but psychological trauma. Alternatively, it must be admitted that the intellect is probably represented throughout the brain and therefore may be affected in cases of widespread neuronal destruction

Of particular interest are those subtle dispositional changes which are noticeable only to close relations Often they are so slight that it is unlikely that they can be due to contusion of the thalamus or to the centres of rage which are situated in the central basal ganglia. Diffuse degenerative lesions of the cerebrum of vascular origin are commonly associated with emotional break-downs extremely distressing to the observer. By analogy it might, with reason, be presumed that like symptoms may be caused by the diffuse lesions of trauma. Also, emotional changes are often most pronounced in cases of aphasia, which suggests that emotion may have the same representation in the cortex as speech. Alternatively it may be argued that dysphasia more than any other disability is apt to lead to emotional breakdowns

Retrograde amnesia, in my experience, has only occurred in those patients who were rendered unconscious immediately on the receipt of trauma and who remained in this state for more than an hour or so. In other words, it occurred in those patients who had suffered the type of accident that causes structural neuronal damage and not in those who merely received

[1] Mitchell, S W "The Relations of Pain to Weather being a Study of the Natural History of a Case of Traumatic Neuralgia" *Amer Jour Med Sc*, 1877, 73, 305

a severe mental shock or a neuronal injury of physiological dimensions When it does occur it is permanent, and the memory for past events never returns. Often a patient will reconstruct what has happened from knowledge that comes to him later, believing that his memory has returned, but it is wise never to accept such evidence as reliable. The events occurring at the time of accident never enter the memory

Grotesque co ordinations of movement so commonly seen in the post-concussional syndrome cannot be ascribed to any known kind of injury or to any neurophysiological mechanism which is capable of producing such locomotor disorders Presumably, therefore they are functional in origin. The same argument also applies to shaking and trembling of the whole body Fine tremors, the result of post traumatic thyrotoxicosis sometimes occur In the latter cases the diagnosis is usually obvious because the patient loses weight, the pulse is fast, sugar appears in the urine and exophthalmos may develop On the other hand, post-concussional functional states rarely lead to loss of weight or to other signs of disordered metabolism

Progressive degenerative encephalopathies rarely give rise to difficulties in diagnosis, as they are usually accompanied by frank and diffuse neurological signs such as increasing spasticity, mental deterioration, deafness and optic atrophy

Although in the present state of our knowledge any analysis of what is functional and what is organic can only be done with some misgiving an attempt must be made in order to establish the premises of the final argument It must be realised however that the difficulties do not end here for even if the above data could be assembled with accuracy the problem of diagnosis would still remain complex, because the patient's final state cannot be the sum of the separate influences of the organic and functional but must result from a complicated integration of both. For example, the abnormal and unpleasant sensations arising from circulatory influences must affect adversely any neurotic state which may result purely from the mental shock of the accident and tend to perpetuate it Alternatively, the unpleasant element of sensations resulting from contusions, etc., will be intensified by a neurotic receptive state

It is well to realise that complexities do exist and to face up to them from the outset. Atypical syndromes which are difficult to understand are apt to be dubbed psychogenic for the good reason that no other explanation can be thought of Such deductions, of course are unsound For example what is it that accounts for a patient's willingness after a severe head injury to live on an inferior level socially and economically ?

Essentially it is a state of mind that allows him to do so, but such a way of thinking may possibly result directly from a complex physical cerebral state. As Straus and Savitsky [1] have pointed out, it is interesting to read accounts by physicians of their own symptoms following injuries to their heads.[2,3] Such people, though originally firm believers in the psychogenic influences in producing symptoms, become converted to the belief that organic changes are really the cause of the post-concussional syndrome. The great argument of the psychogenic theorists is that a patient suffering from the effects of a severe head injury can be rapidly improved by adequate or liberal compensation. The answer to this is, in fact, quite a simple one. Compensation allows a man to readjust himself more easily than one who has not been compensated, and relieves him of family and economic worries at a time of stress. Surely a man's mental state must deteriorate if, because of his illness, his family becomes impoverished and has to accept charity to subsist. Recently I have had the opportunity to go into the homes and talk with the relatives of people injured many years ago There the results of accident can be seen in their true perspective. The opinion of the injured themselves and their families is always the same Even when generously compensated, the injuries have always had far-reaching and sad results. The husband has lost his position in the home, his wife has had to shoulder the burden, and both would give as much as they have received if the accident need not have happened To try and put these simple statements into more scientific language would destroy their genuineness.

In the ability of readjustment, the personal element and background is of course a dominant factor. There is the thrustful ambitious young man who, just beginning to make a success of his career, feels that he must get back to work even though this be done at the price of considerable physical discomfort. Characteristically, a young athlete by the urge of the game will patch himself up to turn out to play at the risk of doing himself serious harm. It is doubtful, on the other hand, whether with the same physical disability he would be willing or able to stand up to the stress and strain of a long route march for which he had no enthusiasm. It is true that it is a very serious injury which completely incapacitates a man, and such injuries are rare. For example, a professional man will carry out a long day's work

[1] Straus, I, and Savitsky, N "Head Injury Neurologic and Psychiatric Aspects" *Arch Neur and Psych*, 1934, **31**, 893

[2] Durand-Weaver, M "Die Commotio Cerebri und ihre Bewertung" *München Med Wchnschr*, 1929, **76**, 1879

[3] Mayer "Die Commotio Cerebri und ihre Bewertung" *München Med Wchnschr*, 1929, **76**, 2135

in spite of a severe attack of migraine On the other hand it would be unnatural for a man to readjust himself in such a way if his hurts were due to other people's carelessness and if his chances in life were such that ambition were virtually non-existent.

MALINGERING

By malingering[1] is meant the deliberate simulation of symptoms and signs for material gain Such behaviour is, of course, not only reprehensible but unlawful and subject to heavy punishment. Fortunately it is rare Exaggeration, on the other hand is common, and in my opinion justifiable in the large majority of cases When there is an obvious physical disability it is unnecessary, but how else can a man whose ailments are entirely subjective declare to those around him how he feels other than by persistent complaining and by demeanour and attitudinisation Hysteria, in common parlance, denotes purely an emotional outburst and must not be confused with rare and ill understood hysterical phenomena Following physical trauma, hysterical disorders almost invariably occur in the form of an inhibition, not as an over action of function A paralysis or a fixed deformity of a limb is the commonest form of the disorder, although it may assume any guise cases of hysterical anaesthesia, blindness, aphasia and sneezing being included in my records It is not regarded as a true form of malingering because it is believed that it is not entirely under the control of the volition, being dependent on actual disarrangement or dissociation of the neural mechanism concerned which explains why fixed postures can be maintained without fatigue Moreover, the mental attitude of the patient is such as to give the impression that he is pleased with his disability and courts rather than avoids interest on the part of the examiner

The Detection of Malingering—The true malingerer often comes under suspicion at the first interview because of obvious lack of frankness and sly looks Unlike the genuine case he takes an intense interest in what is going on around him, although he tries hard to disguise this fact. When asked a question he thinks out carefully what he is going to say before giving his answer which is often in the form of a reasoned argument rather than of the more usual careless and imperfect reply

Also there is a marked willingness for him to theorise on the cause of his illness in a way that is not met with in more genuine cases. During his physical examination he is slow to co-operate

[1] Collie J "Malingering and Feigning Sickness," Longman Green & Co. New York, 1917

rather than awkward or inattentive. Also, he tends to exhibit his feelings by grotesque contortions of his face, particularly when he claims that he is being hurt. Occasionally his attempts at stooping or bending are ridiculous When pretending to move a painful or stiff joint the antagonist as well as the agonist group of muscles go into spasm. Sometimes he makes the mistake of complaining of some specific disability, such as paralysis, anæsthesia, deafness, diplopia or blindness, the simulation of which is easy to detect. In these cases, if he does not trip up when his attention is engaged on some other subject he is almost sure to be caught out when special tests are applied. The number of ingenious tests that have been designed for detection of the malingerer is legion. A few only will be discussed here.

Paralyses.—These do not conform to any known type and are not accompanied by characteristic reflex changes. When, for example, it is claimed that the leg is weak, the knee jerks are often exaggerated, but the Babinski response is flexor. If the patient knows to point the big toe upwards when the sole of the foot is stroked, the other toes fail to fan or spread outwards. Perhaps the most reliable evidence of simulation of spastic paralysis is the manner in which the muscles react to stretch. On attempting to flex or extend a joint, the movements of which are restricted by spasticity of pathological origin, the muscles will slowly then suddenly relax, so that the resulting movement is jerky. In cases of malingering, the greater the force applied the greater is the resistance put up by the patient, as the force is increased he brings other muscle groups into play to give him purchase.

Blindness.—Following closed head injuries, total blindness, whether genuine or otherwise, is exceedingly rare, and in the many thousands of records to which I have had access it occurred only on two occasions. Alternatively, general deterioration of vision is a common complaint. Usually this is genuine, for though it may obviously be due to non-traumatic conditions such as refractive errors, it is introspection consequent on ill-health that leads to the discovery of hitherto unsuspected physical imperfections. In such cases a patient should not be stamped as a malingerer; rather his complaint should be reviewed in the above perspective.

It is the complaint of complete unilateral blindness of which the examiner should be chary. Occasionally the amblyopia of a squinting eye will pass unnoticed until trauma calls attention to its true state, and the patient may claim that his blindness resulted from the accident. Usually the true diagnosis is a simple matter;

a gross refractive error is present and there is a history of long standing squint In my experience unilateral blindness has never been associated with strabismus and there has always been ophthalmic evidence of optic atrophy Absence of pathological retinal change is suggestive of malingering To confirm the genuineness of unilateral blindness the following test will be found useful With both eyes open the patient is asked to read small print at reading distance. When he is doing this a glass prism is placed before the supposedly blind eye In cases of malingering the resulting distortion of vision will interfere with the sight in the good eye so that reading becomes difficult, whereas in the genuine case no such interference occurs Simulated visual field defects may be exposed by chartings made at different distances from the test object, the area of the visual field varying with the distance at which the test is made

Deafness —In bilateral deafness there is usually a characteristic nasal intonation of the voice and an attentive look about the face which is missing in the malingerer When testing it must be remembered that people who are virtually deaf may be startled by loud noises therefore the use of a sudden explosion is not a valid test for the detection of deafness Before deciding on the genuineness or otherwise of a case, wax should be removed from the external auditory meatus and the drum examined If this is not done the examiner will occasionally suffer the annoying experience of someone else making a more accurate diagnosis than he and effecting a cure by simple means In bilateral simulated deafness the examiner has to depend on the patient forgetting he is deaf at all times to the spoken word For example he may obey a command spoken with the ordinary voice when his attention is otherwise engaged Another simple test is ostentatiously to plug the sound ear with a ring of rubber so as to give the impression that hearing on this side has been excluded If under such conditions the patient fails to hear the spoken voice as often he feels he ought then the falsity of his answers is exposed

Commentary —As I feel that it would be wrong to give the impression that the subject of the post concussional syndrome should be approached with a sense of suspicion it has not been my intention to give a complete account of malingering Rather I have attempted to indicate an atmosphere than to make an exhaustive study of the problems concerned particularly as the best results are obtained by conveying to the patient at the outset the impression that you intend to give every side a fair deal and are anxious to make him better Once antagonism has been aroused by the suggestion of partisanship or by impatience, any

therapeutic good that the doctor would otherwise be able to do, either by active treatment or advice, will be nullified. Alternatively, should an obvious inconsistency crop up in the history or clinical examination, then any possible falsity should be probed not only to the patient's but also to the examiner's discomfort.

The greatest difficulty in the assessment of the genuineness of symptoms arises in those cases in which the ailments complained of are entirely of a subjective nature, such as headaches. There is, unfortunately, no scientific means of detecting a malingerer under such circumstances; one's judgment is dependent purely on the general atmosphere of the case, *i.e.*, on one's clinical acumen. Recently more direct evidence has been forthcoming to prove that abnormalities of electrical potential of the brain can be detected in patients suffering from post-concussional sequels.[1] Possibly the solution to this problem will finally be found in electro-encephalography.

It is, of course, very much easier to understand the later complaints of a patient if the acute phases of his illness have been witnessed, for I have often been impressed by the essential similarity of concussional and post-concussional phenomena. For nearly every feature in the post-concussional period there is a corresponding one in the acute phase of the illness, the difference being purely that of intensity. Surely the common sequel to change of temperament must be regarded as the aftermath of the classical stage of irritability.

THE PROBLEMS OF LITIGATION

In cases of litigation the judge in charge of the inquiry will want to know from the doctor:—

1. The nature and severity of the injury complained of.
2. The injured man's present physical and mental state.
3. What the future is likely to show.

As a rule a precise answer can be given to the first question and a satisfactory one to the second. The difficult problem is that of prognosis, and it is with this that the law is chiefly concerned.

Prognosis, of course, is merely another word for prophesy, and in particular the problems to be discussed or settled are:—

1. Will the injured man recover completely, and if so, when?
2. If he will not fully recover, what will be his disabilities?
3. When will he be able to start work?

[1] Williams, D Paper read at the meeting of the Neurosurgical Society, Oxford, July 1941.

4 Will he be able to do his own work, and when?
5 If he is unable to do his own work, what kind of work will he be capable of?
6 What kind of medical treatment ought he to have, and what expense will this entail?

The doctor will find himself ill prepared if he is not ready to answer these questions or at least be able to give a good reason why he is not. He must, of course, never be persuaded to make statements which he cannot substantiate, and must show meticulous care when dealing with facts, otherwise doubt will be thrown on his deductions, and naturally so The law, it must be understood, is essentially out for the truth and nothing else, and the doctor must always work from this premise

When fortified with all the available data on the nature and severity of the injury and with up to-date neurological findings, a doctor is justified, but not until then, in giving an opinion on prognosis based on his own experience and on those of other observers[1] who have made a special study of the subject

First let us consider the type of case in which there is a discrete injury and six months or more have elapsed since the accident

Loss of Smell.—The resulting disability of loss of smell is obvious, particularly in occupations such as those of chemical workers and cooks. In ordinary human beings it adds another hazard to an already sufficiently jeopardised life inasmuch as a patient cannot detect escapes of gas or recognise unwholesome foods before they enter his mouth. Also as Leigh has pointed out, anosmia is commonly associated with that form of frontal injury which leads to prolonged ill health and often to permanent disability

Loss of Taste.—Complete loss of taste is usually due to irremediable injury of the olfactory pathways Partial loss may be caused by (i) partial olfactory damage (ii) contusion of the cauda tympani and great superficial petrosal nerves and (iii) trigeminal denervation of the mouth The sensations of salt, sour bitter sweet, are conveyed by the chorda tympani and great superficial petrosal nerves, and if these are injured the sense of taste is seriously impaired Taste, as the ordinary person knows it, is a complex stimulus being dependent for its finer discriminations on the background of common sensation supplied by the trigeminus[2] Therefore in whatever way the nerve supply to the

[1] Symonds, C. P "The Effects of Head Injury remaining after One Year" Rapport présenté à l'occasion due VII Congrès International des Accidents et des Maladies du Travail. Bruxelles, Juillet, 1935.
[2] Rowbotham, G F "Observations on the effects of Trigeminal Denervation." *Brain*, 1939 62, 364

mouth and upper pharynx is affected, the sense of taste is interfered with. Apart from loss associated with facial paralysis, impairments of the sense of taste rarely recover completely. Ageusia destroys one of the primary pleasures of life Moreover, as patients are constantly reminded of their disability, they often become introspective and miserable

Diplopia.—Most cases of diplopia clear up within six months of injury, and even after this date the majority recover. In my experience the only cases which remained permanent were those due to deformity of the orbit or to fracture lines cutting across the sphenoidal fissure. Even in doubtful cases it is fair to state that the final prognosis is good.

Permanent diplopia is a serious disability, for although double vision can easily be corrected by shading the affected eye, this results in the loss of binocular vision or the three-dimensional view which is essential for the judgments of speed and distance and for many of the finer types of manipulative work

Aphasia.—Motor aphasia is a very crippling disability not only in itself but in the adverse effect it has on the emotional stability of a patient Often though he can think quite clearly he becomes obsessed with his difficulty, thinking of nothing else. Sensory aphasia is even more serious as it constitutes a frank intellectual impairment.

Following Closed Injuries.—When aphasia is discovered as soon as a patient can co-operate, the underlying pathology is usually a diffuse surface contusion. Given that depressed fractures were raised immediately or clots on the surface of the brain removed early, the prognosis is good. When reviewing these cases it is useful to remember that the ultimate recovery of nervous tissue from an injury takes any time up to two years. Therefore when prognosis is in doubt, a final opinion should be withheld until this time has elapsed.

As a generalisation it may be said that by following correct surgical procedure, improvement in most cases of aphasia is likely if special training can be given to the patient afterwards. This usually entails a considerable financial expense which should be considered in the final assessment of damages. When aphasia develops after an interval it is most commonly due to thrombosis and becomes worse rather than better. Theoretically it may be caused by a subdural hæmatoma or cerebral œdema, but these conditions are rare and can easily be confirmed or corrected by suitable measures.

Following Open Injuries.[1]—Aphasia resulting from laceration

[1] Schiller, F "Aphasia studied in patients with missile wounds" *Jour Neurol Neurosurg and Psych*, 1947, **10**, No 4

and destruction of the brain tissue is permanent. Delayed aphasia may be due to thrombosis, but cases resulting from secondary abscess following osteomyelitis of the skull are now being observed. The prognosis in this latter group depends on the success with which the abscess is drained or removed and on how much permanent damage was done before surgical treatment was started.

Defects in the Visual Field.—The resulting disability of a defect in the visual field depends on its extent and whether or not macular vision is affected. When central vision is intact the danger of a blind peripheral area is that injuring objects may approach or be walked into in a way that would not happen in the ordinary course of events. In particular when driving a motor car the extra danger both to the driver and other people is obvious. When central vision is affected the consequences are much more serious to the patient since reading, writing or close work may become difficult or impossible.

Spastic Paralysis.—The disability of any degree of spastic paralysis of the arm or leg is easy to assess. Often it leads to complete incapacity, and a man has to undergo vocational training for some other and more suitable employment.

Sensory Loss of Cortical Type.—Sensory loss due to injury of the parietal lobe is not only an unpleasant sensation but also leads to locomotor impairments. The fingers are clumsy, finer movements are impossible and the hand becomes virtually paralysed. The foot stabs the ground if the movements of the legs are not under the control of the eyes, and walking in the dark becomes dangerous.

In other words, sensory loss of the cortical type in a limb has the same results to a lesser degree as spastic paralysis. Damages may be given on this basis.

Defects in the Skull.—A defect in the skull of reasonable dimensions associated with an intact dura mater and in the absence of infection is not a serious disability, since it may be repaired by simple surgical means which offer excellent chances of an adequate cure. In wounds, however, that are not already soundly healed or that did not heal by first intention, the outlook is quite different. Reopening of the wound may activate latent infection lurking in the deeper tissues which nullifies any attempt at grafting and which, in fact, may leave the tissues in a worse state than before.

When overlying a dural defect a hole in the skull is always serious because operative repair involves considerable interference with the surface of the brain. The importance of a simple defect in the skull is obvious. In sedentary and most other forms of life the brain can be protected from danger by an external shield, the

patient being able to carry on in the usual way. On the other hand, without operation athletes are precluded from playing the more vigorous forms of games. Whether affecting professional or amateur, the results may be far-reaching either from the financial or psychological point of view. Also, a man with a hole in his head finds it much more difficult to get employment than one who is not so afflicted. Superimposed calvarial and dural defects commonly lead to partial but permanent incapacitation.

Symptoms unassociated with Neurological Signs.—The second and larger group is made up of those patients who complain of headaches, dizziness, nervousness, irritability, loss of concentration and insomnia, but who have very little to show for their ailments in the way of physical signs.

This is the most difficult type to assess and the one around which a most heated controversy revolves In previous sections of this chapter attempts have been made to indicate the underlying pathology of each symptom of the post-concussional syndrome and the conclusion arrived at was that the final state of a patient results from a complicated integration of functional and organic influences, each symptom interacting with the others with adverse effects. It has been my experience in the majority of cases that it is useless to start any kind of formal medical or surgical treatment until the problems of litigation have been finally settled. This does not mean, as the representatives of some insurance companies so firmly believe, that their so-called " golden ointment " will cure all ills. It will not. The desire for compensation of a man who has received an injury through no fault of his own is a natural one, and the sooner he is relieved of his sense of hurt the better. Theoretically the assessment of any case would best be made at the end of a man's life, when the effects of the injury could be reviewed in retrospect In practice I believe that problems of litigation should be settled not later than six months after the accident if there are no special medical reasons why this should not be done The arguments in favour of this belief are as follows :—

1. Prolonged worries arising from litigation are avoided.
2. A poor man does not have to live on charity with its demoralising effects.
3. A rich man need not immediately lower his standard of living.
4. The patient can procure those extra comforts in life which he so often thinks are necessary for recovery.
5. He can seek special medical treatment.

6 A man is more likely to readjust himself to his new physical and mental state when thrown on to his own resources than when he regards his state of health as being the responsibility of other people.

7 The attitude of the family changes. Whereas the wife previously sympathised with every complaint and condoned any kind of laziness, she now takes a firmer hand and insists on her husband trying to do something useful and resists his tantrums

When discussing the assessment of a case, not only has the nature of the residual illness to be considered but also the man's normal circumstances and the type of work he will be expected to do It is poor judgment to send a man back to heavy and noisy work amongst machinery if he is suffering from headaches and dizziness Also it is equally useless to advise a business man to return to a post where responsible decisions have to be made if he is nervous and has lost his confidence A special difficulty arises when a man rendered unfit for his own employment is still capable of doing some other kind of work but cannot bear the thought of change and refuses to make the attempt To compel any one other than a young person to do so will result in failure unless prolonged vocational training is given In any case, a change in work not the outcome of one's own desire is usually depressing rather than stimulating if there are no financial or other advantages to be gained thereby Experience has shown that sympathetic and light employment rarely succeeds in getting a man permanently back to work Soon he develops an inferiority complex and feels that his position is unstable which in fact it is The good will of employers not directly responsible for compensation is apt to break down Also a man in sympathetic employment is afraid to exert himself unduly, since he may be regraded cured and lose his partial compensation

Therefore in view of all the above considerations it is better in the long run to make an early and final settlement even of those cases which come under the Workmen's Compensation Act To let civil cases run on for a long time in the hope of recovery is usually a mistake since under these conditions many patients develop further functional complications growing worse rather than better It has been my experience that after severe injuries to the head a man takes about six months after litigation has been settled to readjust himself to his new mental and physical state sufficiently to do useful work and about three years altogether before he is free from symptoms

To neglect medical therapy because the measures are simple is a very grave error in judgment and one that throws patients into the hands of nature curers.

Surgical Treatment—*Lumbar Puncture*.—Before embarking on treatment of post-concussional symptoms the pressure of the cerebrospinal fluid must be measured by spinal manometry, since it is axiomatic that intracranial tension must lie within normal limits if the optimum conditions for the recovery of cerebral tissues are to be established. On those rare occasions when cerebrospinal fluid pressure is high, and there is no obvious intracranial lesion to account for it, spinal drainage or intravenous dehydration associated with restriction of intake of fluids is necessary. When fluid pressure is low and leakage through the nose or ears cannot account for it, fluids in excess of the normal requirements of the body should be taken by mouth. Also, care should be taken to see that adequate amounts and the correct proportions of inorganic salts such as sodium chloride are included in the diet. When symptoms are persistent, repeated manometric tests ought to be made each week.

Encephalography.—The indications for encephalography in the post-concussional syndrome are as follows:—

1. When there are signs of local brain damage, such as spastic paralysis, etc.
2. In cases of persistently raised intracranial pressure.
3. When, in spite of treatment, a patient does not improve.
4. In cases of frank mental deterioration.
5. When the development of air encephalopathy is suspected.
6. For the treatment of adhesions causing traction headaches in an endeavour to break down the adhesions.

Local Explorations.—In cases of persistent and localised pain or the development of secondary headache, a trephine hole should be placed over the painful area and the subdural space explored. By means of a flat metal retractor passed along the space, collections of fluid may be tapped or troublesome adhesions broken down. This is a form of treatment which is far too often neglected, and if the trephine disc is replaced at the end of the proceeding the patient is not left with a defect in his skull.

Subtemporal Decompression.—A subtemporal decompression is rarely needed. In my series it had to be done only in those cases when intracranial pressure remained abnormally high in spite of spinal drainage or dehydration and when the underlying pathological state could not be diagnosed or treated directly. If done indiscriminately as a last measure it will only lead to further complaints rather than to the relief of symptoms. Also it leaves

CHAPTER X

REHABILITATION

REHABILITATION of the head injured consists of a system of graduated mental and physical exercises performed in an atmosphere of encouragement. The physical exercises are formal, the mental tasks informal. Encouragement depends on understanding. In the first edition of this book the following statement was made: "Probably criticisms will be levelled at the rehabilitation schemes that are now being developed by the Emergency Medical Service as being over-elaborate, expensive, and that they cultivate in a patient a taste for hydro life. I believe that time and results will prove these views to be incorrect." Subsequent events have shown that my prognostications have come true. At the beginning of the war organised therapy in the later stages of cerebral trauma was almost unknown; therefore in the first instance method had to be based on first principles and theorising rather than on precept. Now we are able to speak from experience.[1]

TREMENDOUS TRIFLES AND TRIVIALITIES

When life is at hazard, liberty in pawn and acute discomfort taken for granted, it is on little things that cheerfulness and peace of mind depend. Disasters—even blunders—may be forgiven more readily than lack of care or concern for the minor amenities that mean so much to the serving soldier.[2] When health is subnormal, morale at low ebb and anxiety ascendant, it is on the little things that recovery often depends, on the little things that are so easy through familiarity, carelessness or boredom to neglect or to overlook.

For wasted muscles and stiff joints resulting from fractures of long bones, a curriculum of technical exercises has been successfully prescribed by the authorities concerned. By the nature of the problem such clear-cut therapeutic instructions are not possible for the sequels of head injury. Here it is on the little things that the success of rehabilitation depends. In the light of this simple but fundamental principle it is proposed to describe

[1] "Convalescence after Head Injuries." *The Practitioner*, 1946, 156, p. 370.
[2] *Manchester Guardian*, 28th February 1944.

a defect in the skull which the patient can use as a lever for the extraction of compensation.

Defects in the Skull.—When associated with symptoms either of organic or psychological origin a simple defect in the skull should be repaired without hesitation

Eighth-nerve Section.—Eighth-nerve section in the posterior fossa is an operation of considerable magnitude, being necessary only on the rarest occasions. Its indications are frequent attacks of severe vertigo of proved labyrinthine origin which cannot be controlled by conservative measures.

Plastic Operations.—It is wrong to belittle a deformity which is obviously worrying a patient Crooked noses should be straightened, deflected septa resected and ugly scars excised and repaired.

Psychiatrical Therapy.—Much can be done for a neurotic patient without the aid of formal psychiatrical methods if his doctor is interested and willing to spend time in helping him to solve his problems.

When a background of neurosis previous to accident is suspected, but the reason for it is vague, then a patient should immediately be passed over to a psychiatrist No doubt as the need becomes more widely recognised, psychological overhauls will become a routine in the investigation of the post-concussional syndrome in peace as well as in war time.

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[1] "Convalescence after Head Injury." *The Practitioner* 1946, 156, p. 370.
[2] *Manchester Guardian*, 8th February 1941.

in some detail what has been and is being done in one rehabilitation centre serving a large E M.S region

THE CENTRE[1]

This Rehabilitation Centre lies forty miles to the north-west of the combined neurological and neurosurgical clinics, it lies



FIG 235
The object of rehabilitation is to return patients to good health

in a valley of the Cheviot hills of Northumberland, the main peak of the range the Cheviot itself, standing in the north background of the landscape. The Centre is housed in the Castle of Callaly.[2]

[1] Rehabilitation has been so successful that I am leaving this chapter intact, to serve as a model for all such centres which may be established in the future

[2] Callaly was the holding of a Saxon dreng The castle is one of the ancient country houses of Northumbria, having been built some time in the eleventh century It belonged to the family of Callaly until the reign of Henry III, then, owing to financial difficulties, the lands, together with those of Yetlington, had to be sold to Robert fitz Roger fitz John, who later took the name of Clavering The Claverings lived at Callaly until the reign of Victoria They were a well-known Roman Catholic family who took an active part in the national political movements over the centuries and were, of course, ardent Jacobites In the lovely ballroom, decorated in the Chippendale manner, there is much skilfully executed Italian plaster work, busts of kings and prominent people adorn the walls, but two empty frames have been left, these were reserved for the busts of Bonnie Prince Charlie should he survive and return as king In 1877 the house came into the hands of the family of the present owner—Captain Alexander Simon Cadogan Browne The castle is not convenient to run as a hospital but, on the other hand, it is spacious, and the rooms, particularly the dining-hall with its panelled walls, escutcheons, music balcony and set-in fireplaces, have that type of dignity which gives pleasure Finally, the castle has its atmosphere

Callaly Castle stands on the height,
Up i' the day—doon i' the night
If ye build it on Shepherds Shaw,
Then it'll stand and never fa'

The rhyme concerns a legend about the Devil preventing the erection of a building in

To the south rises the precipitous mound of the well wooded Castle Hill. In the grounds are spacious lawns and stately trees and walled gardens with a stream running through from the north west. The stables are elaborate and roomy, and these could be used for special forms of treatment if deemed necessary or for use in cold or wet weather. The open country and beautiful scenery give those facilities which are requisite for recovery of mental and physical health. A pleasant and beneficial route march can for example, be taken in any direction either along the flat valley, over undulating hills, or up precipitous slopes according to the type of exercise thought necessary. The colourings are fresh and varied, and the surroundings in every way are congenial.

THE STAFF

The hospital is administered by the War Organisation of the British Red Cross and the Order of St John. With the exception of two male non-commissioned officers, the staff is entirely female, and are members of the above organisation. The running of the hospital is under the triple control of a commandant, a matron and a medical officer. The commandant, known to her staff as "Madame," is in charge of the Centre, except for the medical and nursing services (Fig 236). The members of the staff who come directly under her control are the cook quartermaster, who holds a diploma in domestic science, an assistant quartermaster and a domestic staff of seven. There is a secretary and an ambulance driver. The matron, who is a fully trained and State-registered nurse, is in entire charge of the general nursing arrangements, and is responsible for the behaviour of the patients (Fig 237). The staff under her control consists of one sister, two assistant nurses and twelve nursing members of the V A D.



Fig 236
The commandant

one place and causing it to be built in another. The modern reading is that a Clavering with due perspicacity and an eye to military safety wished his house to be built on the top of the Castle Hill. His wife, with a deeper and more philosophical sense of the comfort and amenities of living, wished her home to be on the flat of the valley. At night time the supporters of my lady would ascend the hill, demolish the efforts of the stone masons of my lord and carry the stones down to the more convenient lowland site. Needless to say, gravity finally decided the issue.

Of the two non-commissioned officers, one is a skilled physical training instructor and the other is responsible for the military



FIG 237
Matron and her quartermasters

clerical work. These men come under the control of a military registrar, who visits the hospital each month.

The patients are, as in hospitals of similar status in the country, composed of men from the ranks of the various Services; there is no accommodation for commissioned officers In exceptional cases, however, civilian patients can be admitted. The work of the hospital is not confined to neurosurgical problems, on the other hand, all cases at the Head Centre requiring rehabilitation are sent there before final disposal. Details of treatment are laid down by the Head Centre.

PROGRAMME OF THE DAY

The patient's days are well planned and a detailed timetable is given below :—

0630	hrs	Réveillé
0730	,,	Breakfast
0800	,,	Tidying of wards, etc
0900	,,	Muster parade of all "up-patients" Maintenance of hospital grounds, log-cutting and any odd jobs required in the hospital
1030	,,	"Break" until 1100 hrs
1100	,,	Remedial P T exercises
1200	,,	Dinner.
1230	,,	Rest.
1400	,,	Remedial P T exercises or graded games
1530	,,	Tea
1600	,,	Recreation Occupational therapy
1930	,,	Supper
2015	,,	Lectures Games Entertainments
2130	,,	Lights out

Physical training should begin immediately a patient is allowed out of bed It is important that in the convalescent phase patients should not be allowed to develop slovenly habits—for example, they should be instructed to dress properly and not be permitted to shuffle about in slippers or without collar and tie. It is also very easy for a patient to develop a stoop or a limp

After a few days' rehabilitation, consisting of correct dressing, walking and sitting in the ward of the Head Centre, a patient is



Fig 238
Toning up the head injured.

allowed to go into the grounds and after a week or ten days he is transferred to the formal Rehabilitation Centre

For the gradual rehabilitation of patients recovering from head injuries a table of exercises has been compiled, emphasis being laid on balancing movements and re-education in correct walking These exercises consist of trunk bending downwards and sideways trunk rolling in standing and sitting positions, knee raising to chest in lying position, crawling exercises and general physical training exercises (Fig 238) For squints, orthoptic exercises are prescribed patients complaining of dizziness are made to carry out, at increasingly frequent intervals, those movements which precipitate an attack Patients are formed into groups according to the severity of their disabilities, and as they recover they are given stiffer training until they are judged to be fit to return to

duty. If a patient does not progress as rapidly as is expected, he is returned to the Head Centre for further review and advice. Occupational therapy is encouraged, and patients are also asked to help in wood-sawing, grass-cutting and gardening. Lectures and discussions are held by a warrant officer of the Army Education Corps Varied entertainments, such as whist drives and concerts, are provided in the evenings.

DIET

An example of a typical week's menu is given below .—

Day	Breakfast	Dinner.	Dinner Sweet	Tea.	Supper.
Sunday	Porridge, bacon and potato, marmalade	Roast beef gravy, Yorkshire pudding cabbage, potato	Fruit pie and custard	Bread and butter, cake	Brawn, beetroot, fried potato cheese
Monday	Porridge, sausage	Brown stew potato, mashed turnip	Rice pudding and milk	Bread and butter, scones	Soup, potato, corn flour pudding and jam
Tuesday	Porridge bacon, and potato	Meat pies, brussels sprouts, gravy potato	Steamed canary pudding and custard	Bread and butter, tea buns	Welsh rarebit, potato, sweet biscuits
Wednesday	Porridge, savoury, scrambled egg	Boiled mutton, dumplings, mixed vegetables, potato	Jam pie and custard	Bread and butter, rock cake	Oxford pie, gravy
Thursday	Porridge, bacon, and potato	Broth, potato	Syrup sponge pudding and custard	Bread and butter, cake	Sausage rolls, fried potato, gravy
Friday	Porridge, kippers	Fish, carrots, potato, parsley sauce	Steamed chocolate pudding and custard	Bread and butter ginger buns	Cornish pasties, fried potato gravy
Saturday	Porridge, fishcakes	Liver pie gravy brussels sprouts, potato	Semolina pudding	Bread and butter, shortbread	Stovies, potato and gravy

PSYCHOLOGICAL TREATMENT

Psychological treatment consists of the observance of simple principles. A patient must not be frightened by careless remarks; he must be made to understand the true meaning of his illness, he must be continually reassured that he is going to recover his health, and he must have confidence in his doctor and believe that he will be seen through all and not through just some of his difficulties resulting from or precipitated by his injury. Immediately a patient recovers consciousness he must be reassured, not told that he is lucky to be alive as so commonly happens. It is in this early period that the seeds of doubt are often sown

DON'TS

Don't let a patient recovering from a recent head injury see a patient in traumatic stupor, restlessness or delirium The fewer details he hears about the acute phases of his own illness the better. Don't allow fellow patients to stamp the first impression on his mind with their lurid and flowery descriptions of what they have seen—how often a patient tells another that he fought in his delirium and had to be held down by three or more men.

It is at this stage that relatives can do so much harm. By their anxious tired faces they convey to the patient how seriously ill he has been. *Moreover, as soon as the patient is able to listen,* they bring along their own domestic problems to fan the flames of his apprehension. Grumbles about minor difficulties or annoyances, of course, mean little to a large number of people and are only a form of conversation. To the anxious, and particularly when away from the scenes where they can use their own judgment, such simple worries are apt to take on large proportions and prepare the ground for psychoneurosis.

Don't discuss the details or problems of the illness at the bedside, and don't tell a patient immediately that he has a fracture of the skull. All this is best done later when he has better control of his reasoning faculties and is not so impressionable.

During rehabilitation don't be painstaking one day and impatient the next. Don't, through familiarity or boredom, let one's medical attitude change. If a man's progress or behaviour is not satisfactory, don't snap at him or be sarcastic, take him quietly aside and tell him frankly what you think. There are many other don'ts, but sufficient have been given to make a doctor aware of certain common mistakes and to put him on his guard to avoid them.

REASSURANCE

Repeated, detailed and careful examinations will soon gain the confidence of a patient. There is nothing more he distrusts than rapid medical visits at long intervals with questions half answered over the shoulder as one walks to another bed. Explain to a patient the details of treatment long before he asks. Be able to tell him honestly that you have anticipated *all* his medical needs and have made provision for them—a patient finds this most stimulating. When a patient gets up for the first time, see that he avoids bad habits—he must be made to dress completely and to get up at the correct time in the mornings and to fit in with a reasonable routine. If, for example, there has been an undisplaced linear fracture of the skull, it should be explained how unimportant it is. Be firmly confident regarding prognosis. The relatives should be interviewed and the medical problem explained to them. Ascertain whether they are in financial difficulties—advise them if they are. An injured man must be reassured about his wife and family before he can readjust himself, the family also must be reassured if they a[illegible] of anxiety to the patient.

SELF-RESPECT AND DIGNITY

Self-respect is essential not only to happiness but to correct adjustment in society. A man must feel that he influences at least his immediate environment. If he does not, then he may become so demoralised that neither argument nor precept can influence him. Dignity is the bulwark of self-respect. If a man has the opportunity and is accustomed to living nicely in his private life, when crisis arises he will react differently from the man who does not know the good things of life. A reasonable standard of living must be provided at a Rehabilitation Centre and a reasonable code of manners expected in return

How demoralising it is to have one's last meal of the day at four o'clock in the afternoon, composed of bread and butter, plus whatever piquant chance provides, washed down with a pot of indifferent tea How stimulating to hyperacidity and dyspepsia, how long the evening drags on *A properly cooked evening meal at seven o'clock or later, I believe, is an essential part of rehabilitation* It breaks up the day in the right way; it avoids that weariness which so often comes on in the late afternoon when a convalescent patient feels that there is nothing to look forward to that day. A meal makes the evening pleasant and time passes quickly Why have so many convalescent homes been a failure in the past? I believe it is chiefly because of parsimonious catering and ill-cooked food, and to a lesser extent to inadequately warmed recreation and sitting rooms. Correction of these faults is expensive, but why "spoil the ship for a ha'p'orth of tar."

SUBTLETIES

We now come to the subtleties of rehabilitation These are provided by the commandant and the matron. It is not easy to get a man of these islands to talk about his family Yet, until this has been achieved, the last barrier of reserve has not been broken down, behind which may lurk secret and complex problems Before full help can be given to any man he must be persuaded to be frank. How difficult it is if he expresses a grievance in the terms of a headache or a fear as a dizziness How difficult it is for a man to say he dislikes his job or is afraid to return to it Subtlety and a sincere desire to help are the only certain keys to honest expression. How the attitude of a man can change under the influence of the right person. Difficulties of all kinds can easily be solved by the right type of authority.

DISPOSAL

As stated earlier, the periods of rehabilitation vary with the severity of the injury When a man is judged to be fit for duty, he is returned direct from the Centre to a unit Naval and Air Force personnel are always returned to their respective authorities for disposal In the case of Army personnel however a man may first be transferred to a Military Convalescent Depot for further hardening before final posting to a unit

In the case of civilians, a review should be made of progress at the end of, say, a month, and a decision made whether a further course of treatment is necessary or whether the patient is fit to return home and to work When he is discharged he should not be lost sight of Ideally, each patient should be followed into his home by the representative of some society interested in this work to see that, as far as is possible the right conditions are obtained Obviously it is wrong to send a relatively ill woman home to take care of a large family of young children without some kind of help A patient, on the other hand must not be taught to regard herself as an invalid and the people around her must be told not to treat her as such In the case of a working man, efforts must be made to find him the right kind of employment and to give him the assurance that attempts on his part to work will not necessarily result in his being written off as completely fit at the whim of some irresponsible person In civil cases, when a patient has received a large sum of money in compensation for his injury, it would often be better if the money were kept in court, being dispensed as a responsible body thought fit. In the case of children, the money should be spent in bringing them up to the standard of education from which they have fallen as a result of accident Older people often realise too late when most of their money has disappeared that the success of so called 'small' businesses such as newsagents and sweet shops entail long hours of work and diligence

WELFARE SERVICES

Comprehensive welfare services are as important as Rehabilitation Centres Without them final readjustment which means success will in many cases be missing

REHABILITATION IN INDUSTRY

Scientific rehabilitation in industry for the injured it is to be hoped, will be one of the great advances in the post war period

To make this possible new legislation will probably be necessary. After a suitable period of medical rehabilitation a man's physical, intellectual and emotional status must be carefully assessed and agreed upon. A formal medical statement should then be issued and someone made responsible for finding the man suitable employment, the object being to get him back to his original job. The sooner he ceases to be an " odd lot " the better. Given adequate treatment in the acute and convalescent stages of a head injury, few men should remain permanently incapacitated for any kind of useful employment. As stated before, the man must be relieved of anxieties regarding ways and means of living

VOCATIONAL THERAPY

Vocational therapy means the training of a man for some kind of employment within the capacity of his physical disabilities when he has been rendered unfit for his own work. Such training is rarely necessary following the closed type of head injury, and has only to be carried out in the type of injury which results in a frank physical defect Frank physical defects commonly result from penetrating injuries, and no doubt the casualties from European battlefields will make the problem more obvious and urgent. In these cases special apparatus and such adjuncts as hydrotherapy are often necessary to give the best results. Training a disabled man for a new job is an onerous and expensive task. We must see that it is well done.

THE FUTURE

What of the future? Are the Rehabilitation Centres, as we know them, to continue? This is a difficult question to answer. With Service men and civilians the problems are very different. In the case of soldiers and men from other Services, rehabilitation is at least a pleasant escape from the discomforts of training and from the dangers of actual warfare. Moreover the Services are composed of men who are energetic and enthusiastic, also in Service personnel the disturbing influences of relatives has not to be considered. Mothers and wives are only too glad to know that their sons or husbands are safe.

Where civilians are concerned the difficulties are much greater, because a heterogeneous group of patients from a normally energetic boy to a slowly moving, heavily built person of middle or past middle life has to be treated (Fig 239) It is, none the less, always possible to drill people within the limits of their physical capacities if sufficient workers can be employed or persuaded to give their services free. Careful judgment, of course, must be shown in the

choosing of physical tasks, but when this has once been done it is justifiable in the interests of the patient to bring pressure on him to carry them out. This is where relatives begin to interfere, and often demand that the patient be allowed to return home. Moreover a patient may be unwilling in the first instance to go to a Rehabilitation Centre, and may refuse to do so. With care and proper financial support most of the difficulties can be overcome.

RESETTLEMENT IN INDUSTRY

The competent authorities of medicine and industry have now fully realised that the rehabilitation of a person disabled



Fig 239
An awkward squad

In civilian life rehabilitation centres will have to deal with men of differing physiques, emotions and intellects, and not with special groups of men who have been selected for their high physical and mental standards.

by injury or sickness is not solely a medical problem. In fact the link between the final stages of medical treatment and resettlement in the industry is now being forged. Comprehensive study of the resettlement problem has been made and machinery has been established whereby the disabled in search of work can seek advice, guidance and help from the Ministry of Labour.

When restoration in the medical sense has been achieved the services of the social and industrial expert are required: *first*, to determine in consultation with the medical experts whether the patient so restored can return to his previous occupation, and if not what other type of occupation would be most suitable; and *second*, to ensure as far as possible that the restored capacity is used to the best advantage in the field of productive effort,

whether in the previous or in some other occupation During the process of rehabilitation in this wider sense there is a transfer of responsibility from the medical to the industrial services, and the industrial service should begin to operate before the medical service ends. This means that there should be the fullest co-operation between the two services throughout the rehabilitation process." [1]

The final object of the Ministry of Labour is not only to help a disabled man to find suitable employment but to enable him to retain it when once it is found and thereby eliminate so-called sympathetic and philanthropic employment As stated previously, a man must cease to be an "odd lot" if he is not finally to become demoralised.

Ninety per cent. (or 150,222) of the men and women discharged from the Services have been placed in employment. Twenty-three per cent. have returned to their original employment, 58 per cent. required fresh employment and 77 per cent. required special training to fit them for work. It must be realised, however, that these excellent results have been obtained in war-time, when the incentive and opportunities to work are almost ideal. What happens to the disabled after the war regarding their resettlement in industry depends, of course, on international conditions and in particular on the condition of trade. Without special legislation, and this fortunately appears to be forthcoming, the disabled, even if adequately rehabilitated in the medical and in the industrial sense, will scarcely be able to compete favourably with the man who has not been disabled if there is a slump in employment. The genuinely disabled must be carefully provided for in a way that causes them no loss of self-respect or undue hardship. Men must be taught to be willing to change their employment and, if necessary, to make new homes elsewhere. Proper education on this point would make these difficulties easier Also men with abilities above the average must be given ample opportunity to enter the more creative forms of industry. The higher the intellectual and technical standards in industry the better. A man now entering industry should be made to understand that compensation for an unavoidable disability lies in resettlement rather than in monetary rewards which are often unsatisfactory, and often condemn a man to live in virtual poverty.

[1] "Report of Inter-departmental Committee on the Rehabilitation and Resettlement of Disabled Persons" H M S O, 1944 London

CHAPTER XI

POST-TRAUMATIC EPILEPSY

A COMPREHENSIVE definition of epilepsy has not yet been produced and probably will not be forthcoming until the actual nature of the condition is known.

As Kinnier Wilson[1] has pointed out, it is obviously impossible to embrace all its varieties in a single clinical formula, particularly as some are the result of inhibited rather than of excessive nervous activity. The modern tendency is to look upon epileptic phenomena as being the result of an unusual discharge of the neurones either in the form of an overcharge, or of an inhibition, or of a change in rhythm. Whether all epileptic states are essentially the result of pathological processes or of congenital abnormalities within or on the surface of the brain tissue is not known, but what is certain is that structural changes are now being reported rather more frequently than hitherto. This presumably is due to the advance in neuropathology rather than to any change in the etiology of epilepsy itself.

With reason it may be postulated that before epileptic phenomena can develop an epileptic predisposition in some part of the brain tissue occurs which, at intervals, becomes activated by physico-chemical changes within the cells themselves, possibly consequent on circulatory and general metabolic influences.

From clinical and experimental observations it has been clearly established that some brains will go into a state of epilepsy much more easily than others. When epilepsy follows minor forms of closed injury it is believed that the condition has merely been precipitated and not actually caused by the injury, in other words, an epileptic predisposition was already present before the accident. On the other hand, certain forms of injury, such as penetrating wounds associated with sepsis, can lead to epilepsy whether there was a previous tendency in the brain to epilepsy or not.[2]

From the therapeutic point of view it is important to realise that an epileptogenic focus does not necessarily correspond to a pathological focus or to the track of a wound. In fact, wherever

[1] Kinnier Wilson, S. A. "Neurology." London, 1940.
[2] Garland, H. Discussion on Traumatic Epilepsy. *Proc. Roy. Soc. Med.*, 1942, 35, 773.

a structural change due to trauma may be situated, an epileptic attack may start either with psychical, sensory, motor or autonomic phenomena. So-called major and minor attacks are essentially due to the same physio-pathological processes, the difference being determined by the extent of the cerebral disturbances. In minor attacks the abnormal neuronal discharges remain confined to a circumscribed area of the brain, whereas in major attacks the whole brain may be affected. Whether a patient becomes unconscious or not depends largely on the part of the brain concerned but also to some extent on the amount of brain tissue involved. Though epilepsy may not necessarily be caused by a progressive pathological state in the same sense as a degeneration or new growth, repeated attacks may lead to structural changes as a result of the trauma inflicted at convulsive episodes.

PATHOLOGY

An injury to the head does not produce a static pathological lesion within the brain. From the moment of violence, through the stages of phagocytosis to the final consolidation of a wound, or during the phases of possible secondary degeneration or inflammation, the state of the brain is constantly changing. It will be realised, therefore, that epilepsy may result from one or a combination of numerous pathological possibilities. It is surprising, however, how rarely actual solution of continuity of the brain tissue leads to an epileptic seizure. For example, I had a man under my care recently who had been struck over the head with an axe, causing a deep lacerated wound of the motor cortex, and at no time up to his death many days later did he suffer from an epileptic seizure. Epilepsy also rarely occurs during the processes of repair. The condition usually develops some time after repair has been completed and is the result of scar tissue formation or of some pathological sequel such as abscess.

I. Immediate Epilepsy (*occurring whilst the patient is still unconscious*).—This may be due to :—

1. Contusion.
2. Laceration.
3. Hæmorrhage:
   (*a*) Intraventricular.
   (*b*) Intracerebral
   (*c*) Subpial.
   (*d*) Subarachnoid.
   (*e*) Subdural.
   (*f*) Extradural.

4 Bone fragments
    (a) Depressed and causing compression
    (b) Penetrating and irritative
5 Œdema.
6 Hydrocephalus
7 Septic encephalitis.

II Delayed Epilepsy (*occurring at any time up to three months*) — This may be due to —

1 Healing processes.
2 Pathological complications

III Late Epilepsy (*occurring at any time after three months*).— This may be due to —

1 Scars
    (a) Cerebral
    (b) Meningocerebral
2 Cysts, including porencephaly
3 Abscesses.
4 Foreign bodies
5 Chronic subdural hæmatomata
6 Traumatic aneurysms
7 Aerocele
8 Meningitis serosa circumscripta
9 Degenerative encephalitis

As the conditions mentioned in the first two groups have been discussed in previous chapters it is the third group with which we are particularly concerned here, and each pathological state will be described separately

Brain Scars (Cerebral and Meningocerebral).—Brain scars are believed to be the most common cause of epilepsy As they most frequently result from penetrating wounds, and particularly those complicated by the processes of infection the incidence of epilepsy is greater in penetrating than in non penetrating types of injury (Fig 240) Of all the conditions likely to lead to epilepsy, the attachment of the cortex of the brain to the over lying skin consequent on superimposed calvarial and dural defects is the most important. A scar attached to the dura occurs as a greyish gelatinous mass of tough and resilient consistency Histologically a scar is composed of a mixture of fibrous and glial tissue in the meshwork of which are enclosed areas of altered brain tissue The following is a resume of a report given by Professor Shaw on a scar which I excised from the frontal pole of a young boy s brain —

The specimen consists of an oval piece of brain tissue 4 × 4 × 2 2 cm One longer surface is covered by vascular membranes the other is covered

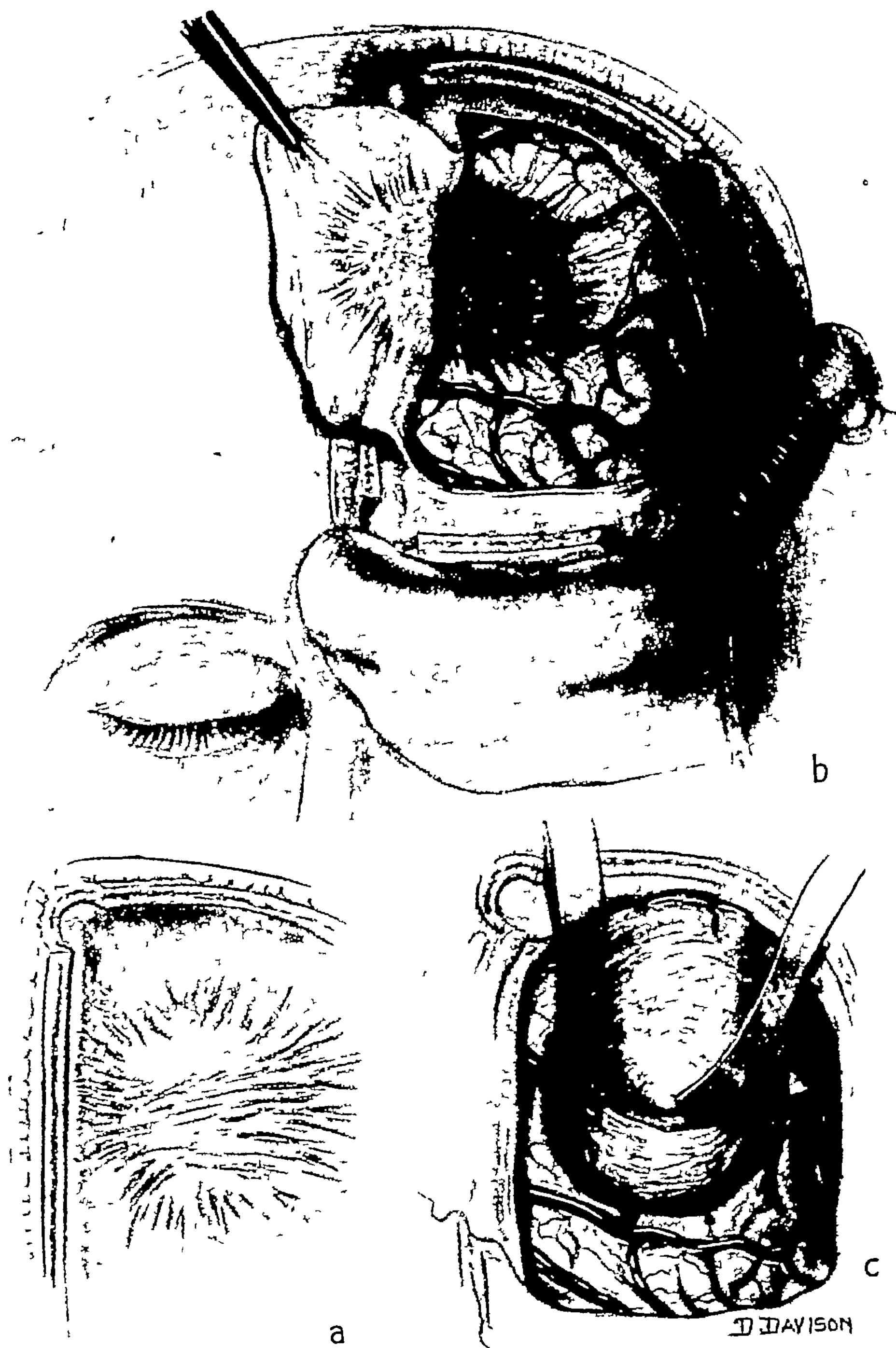


FIG 240

A meningocerebral scar

a, The appearance of the dura mater
b, Reflection of the dura exposing the cerebral scar
c, Removal of a cone of cerebral tissue containing the fibro-glial cicatrix.

by fragmented hæmorrhagic brain tissue and is presumably the line of excision There is a V-shaped depression 2 cm long and 0 8 cm deep on external surface and a section through this shows atrophy and in places disappearance of the cortical grey matter which is replaced by a linear shaped firm pearly tissue like scar tissue extending into the cerebral tissue to a depth of 1 5 cm

Microscopically the surface of the cerebral cortex is covered by a thick layer of collagen tissue of varying grades of maturity in which are a number of arterioles and large venules and also a dural sinus in which is an arachnoid villus with its crest of secretory cells This collagen tissue therefore represents scarring of the dura mater and also probably of the pia-arachnoid as well all of which is adherent to the cortex At the site of the depression the scarred membranes become continuous with a dense

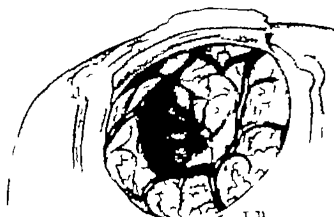


FIG. 41
The dimple of a cerebral scar

scar consisting as special staining shows of collagen exhibiting extreme hyalinosis and containing hæmosiderin this passes down into the white matter and corresponds to the linear scar seen in the gross

Grey matter with its constituent neurones persists on either side of the depression and for some distance on each side there is complete *disappearance of the grey matter* the gap being filled by *extensive gliosis* which extends down into the white matter around the collagen scar In the gliosis are several small irregularly shaped cystic areas

The appearances are indicative of injury resulting in destruction of the grey matter followed by healing due to scarring of the meninges and replacement gliosis The collagen scar deep in the white matter is probably derived from mesoblastic elements of the meninges driven into the cerebral tissue at the time of the trauma

When not attached to the dura a scar is seen as a depression on the surface of the brain the floor of which is composed of shrunken gyri stained by yellow pigment [1] (Fig 241)

[1] Eldridge A R "Brock's Injuries of the Skull, Brain and Spinal Cord," chapter x Baillière Tindall & Cox London, 1940

Foreign Bodies.—A foreign body buried deeply beneath the cortex of the brain is in itself not considered to be an important causative factor in the development of post-traumatic epilepsy. What is important is the track of its wound with its resulting cortical scar (Fig. 212). A fragment of bone which penetrates

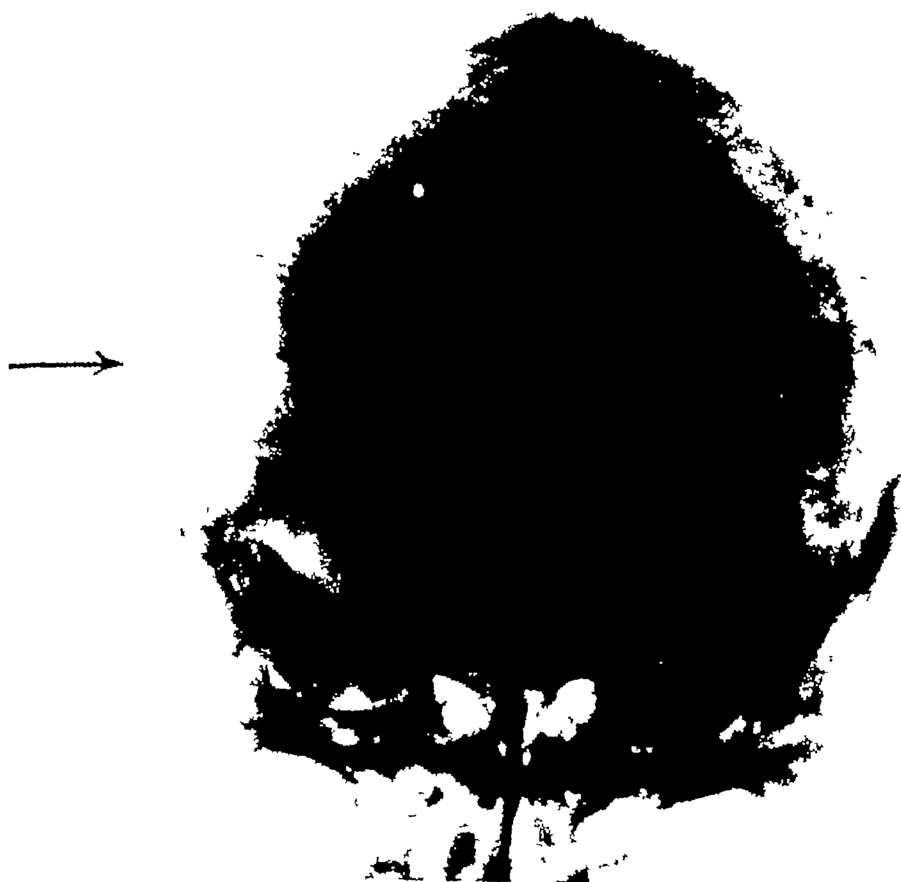


FIG. 212

In penetrating wounds a heavy or large piece of metal often passes deeply into the brain tissue and lodges in the opposite hemisphere. In the superficial parts of the wound track there is often a scatter of bone fragments and small pieces of metal. It is the subsequent meningo-cerebral scar at the entrance wound which is more likely to become epileptogenic.

the dura and which is left spiking the cortex is far more likely to cause epilepsy than when it passes into the deeper cerebral layers, because repeated pulsations of the brain against it are apt to lead to extensive surface scarring. In recent years, owing to enemy action, numbers of patients have received penetrating wounds of the head and have been left with metal fragments embedded in their brain. My opinion is that deeply embedded fragments not removed at the primary débridement should not

be disturbed if they are not causing obvious inflammatory complications, as their removal is most unlikely to lessen the chances of subsequent epilepsy On the other hand, I believe that superficially placed and easily accessible fragments should be removed as a prophylactic measure When epilepsy has already developed,



Fig. 243

Porencephaly In this case a large cavity is communicating with the body of the lateral horn of the right ventricle

bony or metal fragments may or may not have to be removed according to whether or not they lie within the epileptogenic focus

Cysts of the Brain and Porencephaly—Porencephaly implies that there is a hole in the brain and in the true congenital variety ventricles and the subarachnoid spaces are in wide communication. In cases in which the condition is acquired it is usually found in the form of a cystic cavity lined with flattened cells or condensed brain tissue communicating only with a ventricle or with the subarachnoid spaces (Fig 243). Porencephalic cysts often start at the junction of the white and grey matter It is believed that

they result from destructive vascular processes such as hæmorrhage, thrombosis and embolism. Cysts containing yellowish albuminous fluid within the brain tissue or immediately beneath the pia and not in communication with the ventricles or surface spaces are occasionally seen, they also are due to softening following destructive vascular processes.

Chronic Subdural Hæmatomata.—These are often the cause of epileptic seizures, but whether they produce this state by compression or through associated thrombotic changes in the underlying brain tissue is not known. Probably they do so by compression, because drainage of a hæmatoma is usually all that is necessary to rectify the epileptic state.

Abscesses of the Brain are almost invariably the result of penetrating wounds, and occasionally fragments of bone or pieces of metal or cloth are found within their cavities. They are not of common occurrence, but on the other hand they are particularly liable to lead to epilepsy, and this no doubt is due to the fact that they develop hard fibrous capsules which irritate the surrounding brain.

Meningitis serosa circumscripta is the name given to small loculations of cerebrospinal fluid within meningeal adhesions, which result either from direct contusion or to reactions aroused in the lining cells by the irritation of blood in the subarachnoid spaces.

Vascular Anomalies.—In particular, aneurysmal varices or varicose aneurysms are occasionally seen on the surface of the brain when cranial explorations are being made for the treatment of post-traumatic epilepsy. Whether these conditions may result from a blow on the head is extremely doubtful, although theoretically they can. In the large majority of cases they are congenital in origin, and it is known that such conditions may lead to epilepsy at any time in life, irrespective of whether the head is injured or not. It must be admitted, however, that cirsoid aneurysms on the cortex may exist and not produce epileptic phenomena until the adjacent brain tissue has been altered by contusion or other traumatic processes

Aerocele.—As a result of compound fractures of the skull involving the dura, particularly when the paranasal air sinuses are concerned, air may be sucked into the intracranial cavity, to collect either in the ventricles or in the subarachnoid spaces. On occasions a lacerated surface of the brain becomes attached to the margins of a dural fistula, when air may be blown or sucked in the cerebral tissue itself to produce an air cyst. This may increase in size and behave like any other expanding lesion and produce epilepsy, usually of the frontal lobe type, for the good reason that the frontal lobes are usually involved All that is

necessary in these cases in the way of treatment is to repair the causative fistula and to aspirate the cyst.

Neoplasms.—Neoplasms, such as meningiomata, are a common cause of epilepsy, being occasionally found when there is a definite history of severe injury to the head. What part trauma may play in the production of cerebral neoplasia is not known. Until this problem has been satisfactorily settled on a scientific basis it is best to regard new growths following head injuries as coincidental. On the other hand, patients who have had a partial removal of a cerebral tumour and who later receive an injury to their heads commonly retrogress very rapidly and in a way which suggests that trauma may influence the rate of growth of a neoplasm when once it is established.

On rare occasions a cerebral tumour may be the cause of accident, as is well illustrated by the following case. Some years ago I was asked to see a young girl who had received a severe injury to her head, having been knocked from her pedal cycle by a bus. The bus driver stated that the girl was riding unsteadily on her cycle, and that she swerved into him when he was about to pass. When I first saw the girl she was conscious and showed all the signs of a cerebellar tumour including papilloedema. This diagnosis was finally confirmed at autopsy, and inquiry proved that there was evidence of cerebellar dysfunction previous to the accident.

Degenerative and Progressive Encephalopathies.—These conditions, associated with epilepsy, may follow injury, and more will be said on this subject later.

THE INCIDENCE OF POST-TRAUMATIC EPILEPSY

As the following tables will show, the incidence of post-traumatic epilepsy varies considerably according to whether the causative injury was inflicted under peace or war time conditions (Tables I, II and III).

The figures of Ascroft show the results of gunshot wounds of the head in the 1914-18 war, and it is very interesting to note that the incidence of epilepsy is as high as 23 per cent. No doubt the manner in which war time wounds are inflicted—that is, by small objects travelling at great speed and leading to penetration—accounts for the disturbingly high figures. From the 1939-45 war no results on the incidence of post-traumatic epilepsy have yet been published, but in view of the conquest of sepsis the figures are likely to be much lower than those of the 1914-18 war.

Under peace time conditions the mode of injury is different,[1] the head being struck usually by a broad surface so that penetrations and septic complications are uncommon.

[1] Russell, W. R., *Brain*, 1932, 55, 19; *Edin. Med. Jour.*, 1934, 41, 1[illegible]

TABLE I

INCIDENCE OF EPILEPSY IN HEAD INJURIES OF ALL TYPES

Author	Year	Number of Cases Injured.	Percentage Epilepsy	Shortest Period before Onset	Longest Period before Onset.	Remarks.
Allen, Sanford and Dolley [1]	1906	571	4·3			Gunshot wounds. Franco-Prussian War
Behague [2]	1919	3,523	12·1			Gunshot wounds
Sargent [3]	1921	18 000	4·5			Gunshot wounds. Ministry of Pensions.
Rawlings [4]	1922	452	25·0			Gunshot wounds
Allen (quoted by Turner) [5]	1923	167	13·7			Gunshot wounds American Civil War
Steinthal and Nagel [6]	1926	630	28·9		Several years	... /
Wagstaffe [7]	1928	377	9·8	2 months	7½ years	Gunshot wounds.
Steinthal [8]	1929	531	0·2	18 years	18 „	Civilian hospital. Only one case.
Credner [9]	1930	1 990	38·2	Under 1 year	10 „	1 193 of these cases were observed for over 5 years
Feinberg [10]	1934	47 130	0·1			
Marburg [11]	1936		25·0	1 month	23 years	Analysis of several authors.
Elvidge [12]	1940	362	1·03	Three-quarters of an hour	6 months	Civilian hospital Cases observed *for short period only*
Ascroft [13]	1941	317	34·0			Gunshot wounds. 1914 18.
Krause and Schuman (quoted by Marburg)			4·73 to 20·7			War injuries.
Brun (quoted by Steinthal)		470	5·3			War time skull fractures
Reichman (quoted by Steinthal)		603	0·5			Civilian hospital.

TABLE II

INCIDENCE OF EPILEPSY IN HEAD INJURIES WITH PENETRATION OF DURA

Author	Year	Number of Cases Injured	Percentage Epilepsy	Remarks
Behague [2]	1922		Over 12	Periods of onset Shortest—under 3 months Longest—18 months
Rawlings [4]	1922	228	32·8	Gunshot wounds
Steinthal and Nagel [6]	1926	348	31·6	
Wagstaffe [7]	1928	176	18·7	Gunshot wounds with penetration of dura
Credner [9]	1930	1,234	49·5	Periods of onset Shortest—within 1 year Longest—10 years
Ascroft [13]	1941	129	45·0	Gunshot wounds
Brun (quoted by Steinthal)		21	33·3	Gunshot wounds with contusion of motor area

1 Allen, D P, Sanford, L H, and Dolley, D H "Traumatic Defects of the Skull Their Relation to Epilepsy A Clinical and Experimental Study of their Repair" *Bost Med & Surg Jour*, 1906, **1**, 396

2 Behague, P "Etude sur l'Epilepsie traumatique" Thèse de Paris (Arnette, Paris, Editeur) 1919 Analysée par Feindel *Rev Neurol*, 1920, **36**, 88 "Characteristics and Treatment of Traumatic Epilepsy" Questions Neurologique d'Actualité Paris Page 475

3 Sargent, P "Some Observations on Epilepsy" *Brain*, 1921, **44**, 312.

4 Rawlings, L B "The Remote Effects of Gunshot Wounds of the Head" *Brit Jour Surg*, 1922, **10**, 93

5 Turner, J W A "Epilepsy and Gunshot Wounds of the Head" *Jour Neur and Psych*, 1922, **3**, 309

6 Steinthal, K, and Nagel, H "The Capacity for Work after Gunshot Wounds of the Brain, with Special Reference to Traumatic Epilepsy" *Beitr z Klin Chir*, 1926, **137**, 361

7 Wagstaffe, W W "The Incidence of Traumatic Epilepsy after Gunshot Wounds of the Head" *Lancet*, 1928, **218**, 861

8 Steinthal, K "Epilepsy, in Particular Traumatic Epilepsy and the Results of Surgical Treatment" *Ergeb d chir u Orth*, 1929, **22**, 222

References continued at foot of page 413

TABLE III

INCIDENCE OF POST-TRAUMATIC EPILEPSY FOLLOWING INJURIES OF THE HEAD OF THE BLUNT TYPE IN PEACE TIME

Total number of cases injured	430
Total number of cases developing epilepsy	11
Percentage occurrence of epilepsy	2·6

Sex	Date of Injury	Age at Accident	Severity of Accident	[illegible]	[illegible]	Time of Onset of Post. Fit after Accident	Number of fits	Type of Fit	[illegible]	Capacity to Work	Remarks
F	[illegible]	[illegible]	Moderate		No	[illegible]	3	Petit mal	Nil	[illegible]	[illegible]
M	[illegible]	[illegible]	Slight		No	Few hours	Many	Petit mal		Doing well at school	[illegible]
M	[illegible]	[illegible]	Severe	Yes	Yes	[illegible]		[illegible]		Has not worked since	[illegible]
M	[illegible]	[illegible]		?	No	[illegible]		[illegible]		Can only do light work	[illegible]
M	[illegible]	[illegible]		Yes	Yes	[illegible]	Several	Grand mal		[illegible]	[illegible]
M	[illegible]	[illegible]				[illegible]		Petit mal		[illegible]	[illegible]
M	[illegible]	[illegible]	Moderate	[illegible]		[illegible]	Many	[illegible]		[illegible]	[illegible]
M	[illegible]	[illegible]	Slight		No	[illegible]	3	[illegible]		Full work as plumber	[illegible]
M	[illegible]	[illegible]	Severe			[illegible]	Many	[illegible]		Has not worked since	[illegible]
M	[illegible]	[illegible]			No	[illegible]				[illegible]	[illegible]
M	[illegible]	[illegible]				[illegible]				[illegible]	[illegible]

NO —There were no open head injuries in this group.
The expense of this work is covered by generous donation from Miss Needham (Manchester).

As few statements have been made on the incidence of epilepsy following the blunt type of injury I accepted Professor Jefferson's suggestion to make a follow up of a series of cases of head trauma treated in the Manchester and district hospitals It was decided to circularise 1,000 patients whose injuries had occurred not less than five years before This long interval was chosen so that patients developing epilepsy late after an injury would not be omitted from the final figures, which of course was an important consideration as post traumatic epilepsy may develop at any time up to the patient's death. In fact I have a record of seizures occurring twenty years after a fall from the top of a tram In this case local damage was done to the brain, the epileptogenic focus corresponding with the site of the cerebral scar which was in the parietal lobe

Owing to evacuation and other war time conditions, 430 only of 1 000 cases were traceable. This of course, is an obvious imperfection in the compilation of any statistical survey but none the less the figures given above are of value particularly

Credner L. Clinical and Social Effect of Lesions of the Brain. *Zeitsch. f. d. ges. Neurol.* 1930 126, 721.
[illegible] Feinberg. Epilepsie and Trauma 1934
[illegible] Marburg, O. "Die Traumatischen Erkrankungen des Gehirns und Ruckenmark" *Handb. der Neur.* O Bumke and O Foerster 1936, 11, J Springer Berlin
[illegible] Ethridge A. R. "Post traumatic Convulsions and Allied States" Head Injuries of the Skull Brain and Spinal Cord Baillière Tindall & Cox London, 1943.
[illegible] Ascroft, P B. "Traumatic Epilepsy after Gunshot Wounds of the Head" *Brit Med Jour.*, 17th May 1941

as I had opportunity to interview and examine every patient suspected of epilepsy and in many cases to obtain X-rays of their skulls.

CLINICAL FEATURES

Clinically an epileptic seizure may be divided into the following phases :—

1. Prodrome.
2. Aura.
3. Content, or fit proper.
4. Sequels.

By *Prodrome* is meant such symptoms and signs as may occur at varying intervals of hours or days before an attack, and these may be motor, sensory, autonomic or psychical in character Occasionally a seizure is heralded by a change in mood, associated with a feeling of malaise and a fullness in the head. Prodromes, however, are by no means common and are often so unobtrusive as to be unrecognisable except to the most intimate relatives Moreover they are of little diagnostic or surgical value, as they rarely shed any light on the focus or origin of the fit or on the nature of the lesion which causes it.

Aura is the name given to those sensations or motor activities which immediately precede the major event of a seizure and which therefore indicate the part of the brain where the epileptic fit concerned originates. From the surgical point of view an aura is the most important event in any seizure. In fact nothing of value surgically is known about a fit until the absence or presence of an aura has been ascertained, and if present, what its precise character may be.

A fit starting in the autonomic system, possibly in the hypothalamus, usually declares itself by a feeling of uneasiness in the abdomen and by vasomotor changes as shown by circumoral pallor. Sensations of tingling, pins and needles and numbness are referable to the sensory cortex in the post-Rolandic region and can be localised with considerable accuracy. This also applies to motor phenomena when they occur in an orderly manner and slowly enough to permit of visual analysis. Such attacks are usually kn[illegible] cksonian epil[illegible]sy.—Seizures starting in the occipital lo[illegible] ralded by [illegible]llucina-tions of light. Formed [illegible] ly indicat[illegible] visuo-psychic areas anterior to th[illegible] ssure are [illegible] to the in the ears or other hallu[illegible] noise [illegible] [illegible]bes of the [illegible]er str[illegible] on also the temporal [illegible] so-call[illegible] state."

In this condition the patient is not unconscious but is far away from his surroundings and feels that he is a witness of some other world, either of the past or the future. The auras most difficult to analyse and to localise are those which are thought to originate from the frontal areas of the hemispheres of the brain This difficulty is due partly to the fact that frontal auras are so rapid that they cannot be followed by clinical methods, and partly because localisation of function, as we know it, may not exist in the frontal association area. Certainly none has been discovered up to the present time

The sudden cry of a patient about to enter a major fit probably means no more than that the cerebral disturbances are crossing the lower part of the motor cortex and not that the fit has actually started in the cry centre, wherever this might be

Since Hughlings Jackson's work [1] on cortical localisation was confirmed experimentally by Ferrier [2] in Britain and by Fritsch [3] and Hitzig [4] in Germany every part of the cerebral cortex accessible to the surgeon has either been stimulated by electrical currents or surgically excised Much work on this subject has been carried out by Foerster [5] and Penfield [6] and the accompanying diagram embraces the most recent conceptions of the localisation of epileptic auras (Fig 244)

The Fit Proper and Sequels—There are two types of fit—major and minor

In a major fit large areas of the brain are involved in a cerebral disturbance which leads to profound unconsciousness and to a sequence of phenomena that have been so well described by other writers that little need be said about them here According to Gowers there is an aura, usually of an indefinable sensation in the epigastrium, in 50 per cent. of fits of the major type. In a typical attack a patient falls unconscious to the ground uttering a guttural cry, and goes through succeeding stages of tonic and clonic convulsions in which he may bite his tongue, froth at the mouth and pass water He may recover rapidly and carry on as though nothing had happened or he may fall into a deep sleep and be incapacitated by sickness and headache for days Also exhaustion

[1] Hughlings Jackson, J "Selected Writings of John Hughlings Jackson." London, 1931
[2] Ferrier D. "Experimental Researches in Cerebral Physiology and Pathology West Riding Lunatic Asylum Med Rep., 1873, 3, 1-50. "The Croonian Lecture Experiments on the Brain of Monkeys (Second Series)" *Philos. Trans* 1875, 165, 433-488.
[3] Fritsch, G., and Hitzig, E. "Ueber die elektrische Erregbarkeit des Grosshirn" *Arch Anat Physiol wiss. Med.*, 1870, 37 300
[4] Hitzig, E. "Untersuchungen uber das Gehirn" Berlin. A Hirschwald, 1874 8, 276.
[5] Foerster O., and Penfield, W "Epilepsy and the Convulsive State" chapter xix. Baltimore 1931 *Zeitschr f d ges Neur u Psychiat.*, 1930, 30, 47
[6] Penfield, W and Gage L. "Cerebral Localisation of Epileptic Manifestation *Arch. Neur and Psych.*, 1933, 30, 709
Penfield, W and Erickson, T C "Epilepsy" Chas. Thomas, Illinois, U.S.A., 1941

may lead to prolonged paralysis of muscle groups of a limb or limbs.

There are two types of minor fit: petit mal and so-called focal or Jacksonian epilepsy.

Petit mal consists of momentary loss of consciousness or of a disturbance of consciousness short of loss. Typically, a patient will stop what he is doing and stare blankly in front of him for a few moments and then resume in normal sequence what he was doing when the attack intervened.

The essential feature of a focal fit is that a cerebral disturbance begins in a circumscribed focus of the brain tissue and remains

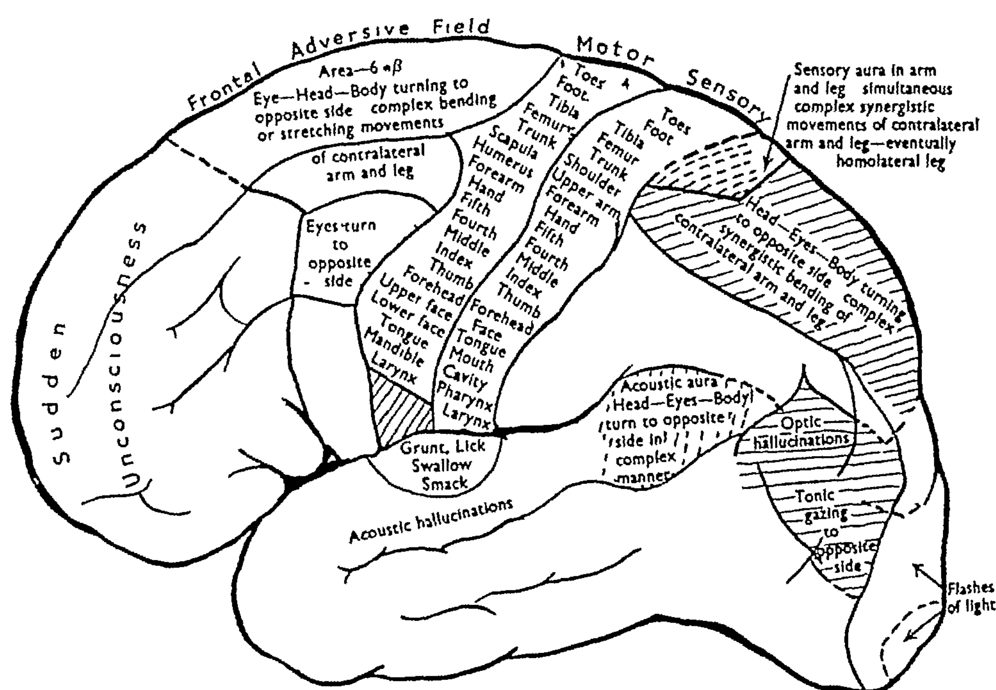


FIG 244
The epileptogenic cortical areas in the human brain

there or travels to other parts of the brain so slowly and in such an orderly anatomical manner that the "march" of the fit can easily be observed. The actual character of a fit depends on the part of the brain concerned and thus may be motor, sensory, including hallucinations of the special senses, or psychical.

Apart from the more stereotyped kinds of epilepsy, there are variants with special features which necessitate particular emphasis, as they occasionally lead to social improprieties, or may even bring a patient within the ambit of the law. Immediately following the major event of an epileptic seizure a patient may enter a state known as "Post-epileptic Automatism." In this condition a patient has full control of his muscle power and

co-ordination, and, apparently, is fully conscious He is not, however, in full control of his judgment, and may perform silly, vulgar or even dangerous acts without being aware of what has happened when finally his automatic state disappears. When fits are of a psychical character all kinds of complicated hallucinations may be experienced which may lead to or be associated with emotional outbursts or acts of violence known as brainstorms One of my patients, a married man, suffering from such attacks would waken in the night with the belief that his late fiancée was entreating him to reconsider his decision and to go home with her This would start a struggle in which he would attempt to evict her from his bedroom in the fear that his wife would waken and discover his embarrassment. So violent would the struggle become that it would end on the floor, and his wife's description of the attack was indistinguishable from that of a convulsive seizure. Apart from these attacks the man was perfectly normal and was fully occupied in useful work.

Occasionally inexplicable head on collisions between motor cars on the open roads may be accounted for by the momentary "black out" of an attack of petit mal or by a transitory post epileptic automatic state. This possibility was graphically brought to my notice recently in the case of a young man who was sent to me suffering from epileptic seizures. A short time before he had crashed a vehicle of a local authority into the front of a house, killing several occupants According to his own evidence the accident was the result of a momentary amnesia and not to the cause which was found by the Coroner's Court. More will be said on this subject when the question of compensation and litigation is considered

Electro-encephalography [1]—As stated in Chapter III oscillations of electrical potential normally occur on the surface of the head at the rate of about ten per second when the eyes are closed and the mind at rest. In normal activities of the brain the Berger rhythm or alpha waves as they are called, may disappear Alterations in character of these waves or loss of their rhythmicity is indicative of abnormal activity within the brain even if this does not manifest itself either as subjective or objective neurological phenomena. Abnormal changes in the electrical waves of the brain always occur during an epileptic seizure. Commonly they are seen just before and occasionally in the interval between fits

A high voltage wave is thought to be due to an excessive discharge of a group of neurones and is known as hyper synchrony Loss of normal rhythm is spoken of as dysrhythmia.

[1] Russell Brain, W "Recent Advances in Neurology" 4th ed. London, 1940.

According to Gibbs, Gibbs and Lennox,[1] grand mal, petit mal and psychomotor attacks have distinct wave formations. In grand mal the waves increase in frequency up to thirty per second and appear as sharp spikes. In petit mal, rhythm is slower than normal and spiked alternately with rounded or flat-topped waves. In psychomotor attacks the rate is slower than normal and the flat-topped waves predominate.

By suitable placing of the electrodes, electro-encephalography can indicate the site of the brain in which the electrical discharge originates. In some cases it can be shown that epileptic attacks may start from a circumscribed area on one side of the brain, from corresponding and symmetrical sites on two sides of the brain or as a diffuse simultaneous discharge[2]

Delta waves are slow and heaving in character and occur at random frequencies. They indicate that some part of the brain in the region from which they emanate is electrically "dead" and therefore diseased. Thus electro-encephalography can not only localise an epileptogenic focus and throw light on the type of seizure that occurs but may also detect and localise a pathological focus (Fig. 245).

Encephalography.[3]—The radiographic visualisation of the ventricular system, basal cisterns and subarachnoid spaces over the cerebral hemispheres, by the replacement of quantities of cerebrospinal fluid with oxygen or air via the lumbar or cisternal route is known as encephalography In order to determine with precision the pathological state of the brain in cases of post-traumatic epilepsy, at least 75 c.c of the chosen gas must be injected. Moreover, positions other than the routine ones are necessary. For example, if there is a filling defect over the frontal lobe of the brain or in the anterior horn of the lateral ventricle, a "nose-up lateral shoot" (Fig. 246) is useful, as this position ensures that the air must collect in the frontal region if pathological obstructions are not present. The "nose-down lateral shoot" (Fig. 247) position is useful when a lesion is suspected or proved to be in the occipital region. It is scarcely necessary to point out that air is lighter than cerebrospinal fluid and collects in the uppermost part of the intracranial cavity. Therefore when other special views are needed the head must be so oriented that the suspected area is uppermost. The opposite, of course, applies when the skull is being examined, since a fractured area is shown in greatest detail

[1] Gibbs, F A, Gibbs, E L, and Lennox, W G "Epilepsy A Paroxysmal Cerebral Dysrhythmia" *Brain,* 1937, **60**, 377
[2] Jasper, H, and Kershman, J "Electroencephalographic Classification of the Epilepsies" *Arch Neur and Psych.*, 1941, **45**, 903
[3] Shanks, S C, Kersley, P, and Twining, E W. "A Textbook of X-ray Diagnosis" Lewis & Co London, 1938-39

when placed nearest to the film Stereoscopic views should be used as a routine procedure, but when this type of examination

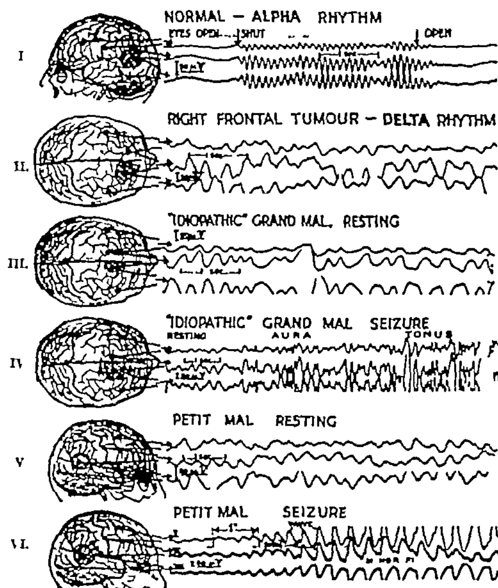


Fig 243

The diagrams on the left show the positions of the leads and of the discharging foci (shaded); the tracings on the right show the corresponding changes of electrical potential.

(*From Brain Recent Advances in Neurology Electro-encephalograms kindly lent by Dr W Grey Walter*)

I Normal alpha rhythm.
II Delta rhythm in a patient with a right frontal tumour
III Resting rhythm in a patient with "idiopathic" grand mal.
IV Seizure rhythm in a patient with "idiopathic" grand mal.
V Resting rhythm in a patient with petit mal.
VI Seizure rhythm in a patient with petit mal.

is not possible views taken in two axes with the head in the same position should be filmed (Fig 248). With careful technique all

gross lesions of the brain and many of lesser degree can be accurately localised and outlined.

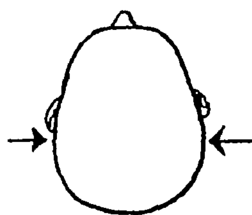


Fig 246
The X-ray position of a "nose-up lateral shoot"

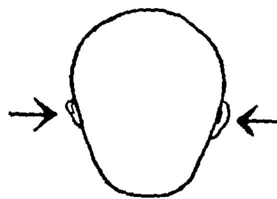


Fig 247
The X-ray position of a "nose-down lateral shoot"

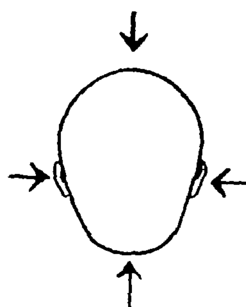


Fig 248
Views taken in two axes

The following is a list of the encephalographic findings which I have obtained in cases of post-traumatic epilepsy (Fig. 249):—

1. Normal ventricular outlines and normal subarachnoid spaces.
2. Normal ventricular outlines with excessive collections of air over the cortex, suggestive of faulty absorption of the cerebrospinal fluid at the arachnoid villi.
3. Normal subarachnoid spaces and bilateral dilatation of the ventricles indicative of obstruction to the circulation of cerebrospinal fluid within, or at the exit of, the ventricles. The obstruction, of course, in these cases could not have been complete, otherwise air would not have entered the ventricles. An alternative explanation of the ventricular dilatation is bilateral cortical atrophy.
4. Unilateral dilatation of a lateral ventricle. This is indicative of a unilateral cortical atrophy. When displacement of the ventricle is also present, this means that the atrophy is associated with a meningocerebral scar.
5. Porencephaly. In these cases a large circumscribed cavity was seen either in communication with the ventricles or with the subarachnoid spaces
6. Local filling defects in the subarachnoid spaces suggestive of meningocerebral scars.[1]
7. Flattening of the lateral ventricular outlines with displacement, suggestive of a space-occupying lesion such as abscess, tumour or subdural hæmatoma.
8. Filling defects in the body of the lateral ventricles without ventricular displacement, suggestive of intracerebral hæmorrhage.

[1] Olivecrona, H "Corticomeningeal Scars in Traumatic Epilepsy" *Arch Neur and Psych*, 1941, **45**, 666

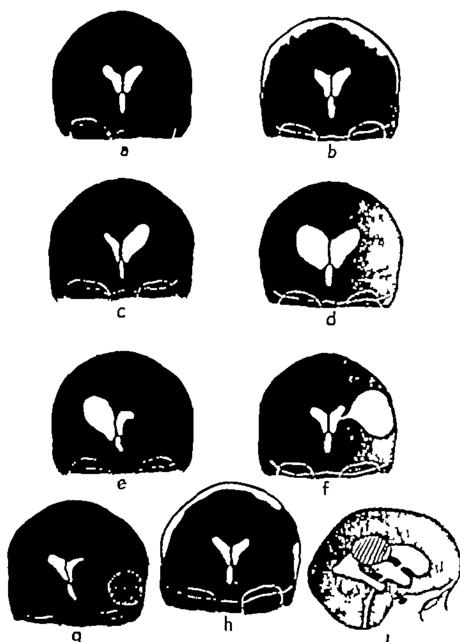


Fig. 240

Encephalographic findings in post traumatic epilepsy

a Normal ventricular outlines.
b Dilated subarachnoid spaces.
c Unilateral hydrocephalus.
d Bilateral hydrocephalus.
e Unilateral hydrocephalus with displacement
f Porencephaly
g Displacement due to abscess.
h Surface-filling defects due to meningocerebral scars
i Filling defects in the lateral ventricle

At the outset it must be realised that encephalography does not and cannot localise an epileptogenic focus which often is

distant from a pathological lesion; neither can it shed any light on the nature, extent or pattern of a seizure. It does, of course, give information on the gross structural state of the brain, and often with much greater precision than any other kind of examination.

TREATMENT

The treatment of post-traumatic epilepsy is largely an economic rather than a medical or surgical problem, particularly as the condition rarely leads to neurosomatic degeneration, in which state a patient becomes mentally impaired and his body deformed by spastic paralysis. Even in severe cases epileptic episodes occupy a very short period of a patient's life and often not more than a few minutes or hours in a year.

Apart, therefore, from a few obviously dangerous occupations, such as car driving, working on scaffoldings or on the edge of vats, a man suffering from occasional epilepsy may live a normal life and must be encouraged to do so. There are dangers, of course, in working amongst machinery, and the best occupation for an epileptic is work on the land. Alternatively, whenever a man is found to be suffering from epilepsy it should be incumbent on the doctor to point out the dangers of driving and to urge him to cease doing so; my experience in the following case will illustrate why. Some years ago a patient of mine received a large sum of money in compensation for epilepsy following an injury to his head. With the proceeds he bought a motor car, and during a seizure crashed into a telegraph pole, killing himself and seriously injuring his passengers

Medical Measures.—A patient suffering from epilepsy may be allowed a normal diet, but should take regular meals so that his blood sugar is never allowed to fall to a low level. When regular feeding is not possible, sweets or barley sugar should be carried and taken on those occasions when something more substantial is not obtainable. Also, sweetened-milk drinks taken before retiring to bed may avoid those epileptic episodes which occur early in the mornings or immediately after getting out of bed. Excessive physical strain, excitement or worry should be avoided. If drugs are supplied they must be given in sufficient quantities to give the desired result, but not in doses which cause toxæmia or drowsiness. A routine which I now adopt is to give $\frac{3}{4}$ gr. epanutin in the morning and $\frac{1}{2}$ gr. luminal at night, these amounts being increased as is found necessary.

Surgical Measures.[1]—For patients whose epileptic episodes are of such severity or frequency in spite of medical measures that

[1] Penfield, W "Epilepsy and Surgical Therapy" *Arch Neur and Psych*, 1936, 36, 449

their means of livelihood and mode of living are seriously affected the necessity of surgical treatment will have to be considered Also, surgery is justified in suitable minor cases when for professional, social or domestic reasons epilepsy of however infrequent occurrence or mild a character, will lead to far reaching *consequences*

The objects of surgical treatment are twofold (1) to remove a pathological lesion which is thought to be the cause of the



FIG. 250

A collection of air in the dimple of a cerebral scar over the frontal lobe

epilepsy, and (2) the destruction of that focus of the brain in which an epileptic seizure originates

Indications for Surgical Treatment—By clinical and electro encephalographic means that part of the brain in which the origin of a fit is suspected is identified (Fig 250). Possibly the two foci localised by these methods do not coincide and a decision has to be made as to which is giving the more reliable information. When an aura develops slowly and permits careful analysis clinical evidence may be accepted and acted upon On the other

hand, when an aura is rapid and precise analysis is not possible, the findings of electro-encephalography must be given preference A pathological focus is determined either by neurological signs or by encephalography Usually a combination of both these methods is necessary. When a pathological lesion coincides with an epileptogenic focus then exploration is indicated without any question. In those cases when a pathological process is distant from the site at which an epileptic seizure starts, it is often difficult to decide whether to operate or not Providing other methods have been given a fair trial and have failed to give relief, I believe that it is justifiable to explore the brain in order to remove the pathological focus and to diathermise the epileptogenic area if this is also feasible through the same exposure.

Operative Technique.—An abscess is removed together with its capsule; a cyst is drained and, if possible, its wall dissected out; a blood clot is evacuated and drained; a surface aneurysm is ligated and a new growth is excised.

As these conditions are not peculiar to trauma, nothing further will be said here on the operative technique of their treatment. The details may be found elsewhere. It is with the excision of cerebral and meningocerebral scars that we are principally concerned.

Exposures are made through osteoplastic flaps, which are so designed that the whole of a pathological lesion together with the epileptogenic focus is uncovered (Figs 251 and 252). When for anatomical reasons this would necessitate an excessively large flap, the exposure may be confined to the region of the pathological change. Operations must be carried out under local anæsthesia; drugs, basal or general anæsthetics not being given as these so damp down the excitability of the cortex that it becomes unresponsive to electrical stimulation.

Let us now suppose that we have exposed the dura mater and that a meningocerebral scar exists. By means of a *U*-shaped incision the dura mater is reflected upwards. Under the circumstances mentioned this will necessitate division of the adhesions between the cortex and the under surface of the dura As meningocerebral adhesions are tough they do not tear easily, and are best divided by a diathermy current. To do this a small metal hook is placed on the far side of an adhesion, and as this is put on the stretch the hook is touched with a diathermy button. After the dura has been reflected the exposed cerebral cortex is carefully inspected. From the pattern of the blood vessels and shape of the gyri it is impossible to delineate the motor cortex, and this must be done by means of electrical stimulation with a faradic or galvanic battery, or best, with a thyratron. Care must be taken

not to use too strong a current, as a major convulsive seizure may thereby be precipitated from any part of the cortex. This is a very dangerous happening if the veins in the region of the parasagittal sinus have been exposed because they may become so engorged with blood as the patient strains that they snap and bleed uncontrollably.

Before going further it must be stressed that no part of the brain subserving a function which is essential to normal life must



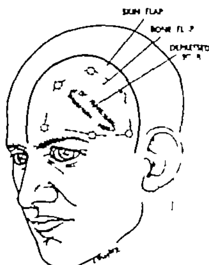


FIG 231

A patient after partial lobectomy for removal of a frontal meningocerebral scar. An osteoplastic flap is the best method of exposing a traumatic focus or scar in the frontal lobe of the brain. A skull defect can quite easily be repaired by a rib graft at the same time as the epileptogenic focus is excised. The fact that the calvarial defect [illegible] the operative bone flap makes no difference to the final consolidation of the rib graft.

FIG 232

The approach to a menin[illegible] scar situated in the frontal [illegible]

be injured during any surgical manipulation. For example, it is obviously wrong to leave a patient aphasic or hemiplegic in an endeavour to cure him of epilepsy.

Let us now suppose that the pathological focus may be excised without damage to important areas of the brain. The line of excision, enclosing if possible the epileptogenic focus, is mapped out and the vessels crossing this line are coagulated by a diathermy current. A convenient way of doing this is by the following method. With a pair of fine non toothed dissecting forceps a hole is made through the pia on each side of the vessel to be coagulated, the vessel then being picked up between the blades of the dissecting forceps and coagulated. Without first making holes in the pia, the toughness of the membrane prevents the vessels from being picked up cleanly. The vessels are then coagulated in like manner half an inch internal to the line of

excision and divided with scissors close to the first point of coagulation. Then with a sharp scalpel the incision is deepened and a block of tissue removed as far as the ventricles if the scar extends as deeply as this. The next step is to stop all bleeding points with meticulous care. On those occasions when the epileptogenic focus has not been removed in the block resection of the pathological process, its position ought again to be determined by electrical stimulation and the area diathermised or excised. Personally I depend on diathermy alone for destruction of an epileptogenic focus distant from a pathological process.

The next procedure is to close or repair the dura if tissue has been lost in such a way that adhesions do not reform between it and the cortex. This is best done by covering the damaged area of the brain with a sheet of amnioplastin on fibrin films[1 2] Defects in the skull when present should also be repaired at the same operation by one of the methods described in Chapter IV. The slightest degree of infection will prevent healing and nullify any good that might result from the operation, and in fact may leave the patient in a worse condition than before. Finally, owing to lack of tension, post-operative clots are particularly liable to occur in these cases, and therefore before a wound is closed every vessel, however small, must be firmly sealed. Anti-convulsive drugs should be given regularly for at least six months following operation, or for as long as may be found necessary.

[1] *Journal of Neurosurgery* January 1944, 23
[2] *Ibid* May 1944, 171

CHAPTER XII

THE FINAL RESULTS OF HEAD INJURIES

THE majority of injuries to the head are in the nature of simple scalp wounds or abrasions Such wounds rarely lead either to complications or to sequels, although a simple blow may occasionally produce an apprehension neurosis, precipitate epilepsy or cause a subdural hæmatoma Simple scalp wounds are as a rule not admitted to the wards of civilian hospitals but are treated in the out patient departments and are not included in statistical surveys of head injuries On the other hand simple scalp wounds find their way into the beds of Service hospitals and considerably dilute the figures of the more severe types of cerebral injury

GUNSHOT WOUNDS OF THE HEAD

On the subject of gunshot wounds of the head a large volume of statistics has accumulated The following series of figures have been chosen to illustrate the prognosis of this type of wound

Cushing's Figures [1]—From 23rd July to 31st October 1917 250 cases of injury to the head came under the care of a surgical team of which Cushing was a member Of these 219 were selected deliberately because of their major gravity The nature of the injuries is listed as follows —

(*a*) 22 cases with scalp wounds (10 1 per cent)
(*b*) 54 cases with wounds involving the cranium as well as the scalp, but with intact dura. (24 6 per cent)
(*c*) 133 cases with wounds with dural penetration (60 7 per cent.)
(*d*) 10 cases with bursting fracture and with very serious intracranial complications
(*e*) 20 cases who were beyond surgical aid on admission
(*f*) 11 cases whose injuries were allowed to pursue their course without actual operation

[1] Cushing H "A Study of a Series of Wounds involving the Brain and its Enveloping Structures. *Brit Journ Surg* 1917 18 5, 558.

The results in the 133 cases in which there was dural penetration were as follows :—

(*a*) In the first consecutive 44 cases the mortality rate was 54 5 per cent.
(*b*) In the second consecutive 44 cases the mortality rate was 40 9 per cent.
(*c*) In the third consecutive 45 cases the mortality rate was 28 8 per cent.

Of 71 post-operative fatalities which occurred in the whole group of the 250 cases .—

12 deaths were due to non-cerebral causes such as pneumonia.
16 deaths were due to severe traumatic cerebral lesions
43 deaths were due to intracranial infections : 14 to meningitis, 12 to ventriculitis, 9 to encephalitis and 8 to abscess.

In the light of previous and contemporary experience in the treatment of cerebral injuries, these figures were not only excellent but they opened a new era in the prognosis of gunshot wounds of the head. Good, however, as Cushing's results were, it must be realised that they were concerned largely with the survival of patients. Many of the patients were left maimed by hemiplegias, etc., the result of destruction of cerebral tissue.

Jefferson's Figures.[1]—

ANALYSIS OF 220 CASES OF GUNSHOT WOUNDS OF THE HEAD

	Scalp Wounds	Fractures of Skull No Dural Injury	Fractures with Dural Penetration
Number of cases not previously operated on	34	37	79
Number of cases operated on elsewhere and transferred to base hospital	20	16	34
Totals	54	53	113

Of the 150 patients not operated on before they came under Jefferson's care 29 died, and in none of the fatalities was the dura

[1] Jefferson, G "The Physiological Pathology of Gunshot Wounds of the Head" *Brit Jour Surg*, 1919-20, 7, 262

intact. The total mortality was 19 3 per cent., the mortality for penetrating wounds of the brain being 37 6 per cent. Of the 29 deaths, 4 were due to brain injury and 25 to infection. The absence of fatalities when the dura was intact emphasises the importance of the dura as a barrier to infection

Jefferson stressed the importance of removing all indriven fragments of bone, as bacteriological examination showed that such fragments were invariably infected both by aerobic and anaerobic organisms Moreover, he was one of the first to point out that diffusion of infection occurred via the ventricles rather than over the cortical spaces

Ascroft's Figures (1943)[1]—

ANALYSIS OF 500 CASES OF HEAD WOUNDS DUE TO MISSILES

Type of Wound.	Mortality in Whole Series.			Disposal of Recovered Patients		
	Patients.	Deaths.	Mortality Per Cent.	Patients.	Invalided on Account of Head Wound.†	To Duty
Scalp wound only	83	0	0	80	1	76 (03°)
Fracture of skull—dura intact	130	2	1·6	130	10	110 (85 ₒ)
Dura pierced	29*	44	15·0	226	93	1*4 ([illegible])
Totals	516	46	9 0	436	104	310 (71)

* Data not available in 34 cases.
† Twenty-two patients fit for duty as far as the head wound was concerned, were unfit on account of other wounds.

Eden s Figures (1944), p 430

Schorstein s Figures (1944), p 431

The final prognosis of disability resulting from serious focal destruction of brain tissue such as commonly occurs in penetrating wounds, is soon obvious. Hemiplegias, monoplegias, aphasias and hemianopias are not likely to improve materially after one year Neurophysiological impairments which are going to do well usually

[1] Ascroft P B. "Treatment of Head Wound due to Missiles: Analysis of 500 Cases. *Lancet* 21st August 1943 211

Eden's Figures (1944).[1]—

Classified Results of Primary Operations

Type of Case	Number of Cases Operated On	Deaths	Healing by First Intention	Infected Wounds*	Complications
Scalp Wounds . .	139	0	135 (97.1%)	4	1 Extradural abscess
Compound Fractures (Dura Intact)—					
Depressed fractures .	45	0	41	4	1 Sequestration
Compound fissured fractures	7	1	5	1	
Sagittal sinus injuries	2	0	2	0	
Air sinus and eye wounds	9	0	5	4	1 Sinusitis 1 Rhinorrhoea
Face wounds . .	1	0	1	0	
Ear wounds .	5	0	4	1	1 Mastoiditis and subcutaneous abscess
Total .	69	1	58 (84.1%)	10	
Open Brain Wounds—					
Perforating brain wounds (metal in-driven)	51	12	35	7	4 Cerebral fungus 4 Meningitis 1 Cerebral abscess 1 Fits 1 Cerebrospinal fluid leak
Compound fractures (in-driven bone only)	24	5	19	4	2 Cerebral fungus 1 Meningitis
Compound fractures (dura torn)	10	0	9	1	2 Fits
Air sinus and eye and brain wounds	7	2	5	1	1 Pulmonary embolus 1 Rhinorrhoea
Ear and brain wounds	3	2	1	0	1 Mastoiditis
Facial brain wounds	1	0	1	0	
Sagittal sinus brain wounds	6	3	3	1	1 Sagittal sinus thrombosis
Total	102	24 (23.6%)	73 (71.6%)	14	
Grand Total .	310	25 (8.1%)	266 (85.8%)	27	

* Including wounds left open

show definite signs of improvement within the first few months after injury and often within the first few weeks.

Late infective complications are by no means uncommon following penetrating cerebral injuries, even when the associated

[1] Eden, K., "Mobile Neurosurgery in Warfare. Experiences in the Eighth Army's Campaign in Cyrenaica, Tripolitania and Tunisia," *Brit. Jour. Surg.*, April 1944, 327

Schorstein's Figures (1944).[1]—

Case	Direction of Missile	Interval between Wounding and Operation.	Type of Operation.	Result
		Hours		
1	L. fronto-orbital	36	Fascial graft	Died
	Midfrontal, R. maxillary	240		Healed
3	R. fronto-temporal	24		
4	L. fronto-orbital	Over 48	Soft paraffin pack	
5	R. fronto-orbital	24		
6	R. naso-L. orbital	Over 24		
7	R. orbit-L. maxillary	Over 24		Not known
8	L. fronto-temporal	Over 24	Dura closed Soft paraffin pack	Healed
9	L. fronto-orbital	24	Superficial débridement *	Died
10	L. fronto-R. orbital	24		"

* Operated on elsewhere

Northfield's Figures (1944).[2]—

Total number of admissions for the three months following "D" Day		193
Of these 61 were penetrating injuries.		
Total number of cases operated on		128
Of these there were 15 deaths.		

McKissock's Figures (1944).[3]—

Total number of admissions for the six months following "D" Day		531
Of these 229 needed further operative treatment.		
There were 93 penetrating injuries.		
Total operative mortality on the 229 cases	4 3 per cent	
Operative mortality on penetrating wounds	7 5 "	

O'Connell's Figures (1944).[4]—

Total number of admissions for the two months following "D" Day		180
Of these 67 were penetrating wounds.		
132 cases were operated on.		
Operative mortality on 58 penetrating wounds	7·0 per cent.	

scalp wound has firmly healed by first intention and the patient is apparently well. Spreading suppurative encephalitis, when it does supervene, usually takes place within the first year following wounding but cerebral abscesses may form at any time up to twenty or more years after injury

Occasionally a progressive degenerative encephalopathy supervenes after a cerebral wound has remained healed, or at least quiescent, for many years.

[1] Schorstein, J "Gunshot Wounds of the Fronto-orbital Region." *Lancet* 8th January 1944 44

[2-4] Personal Communication.

The incidence of epilepsy following gunshot wounds of the head received in the 1914-18 war was extremely high. According to Ascroft's figures,[1] and these were based on 317 cases, 107 or 34 per cent. developed epilepsy some time after injury. The incidence was as high as 45 per cent. in those cases where there was dural penetration. In skull and scalp wounds where the dura was intact the incidence of epilepsy was 23 per cent Possibly after this war, owing to the better control of sepsis, the incidence of epilepsy following penetrating wounds will be materially reduced.

BLUNT INJURIES OF THE HEAD

The prognosis of blunt injuries to the head depends on three factors: on the type of man injured, on the nature of injury and on the circumstances which obtain following injury.

Type of Man Injured.—A man may have a strong, well-formed body with a sound digestion and a good resistance to disease. On the other hand, he may be muscularly weak and ill-formed and may suffer from constant physical discomfort, such as dyspepsia or rheumatism.

A man born of stable parents may inherit a sound emotional constitution; alternatively, a man may have psychoneurotic parents and may show similar tendencies in ordinary life Many people of apparently sound emotional constitution may, in fact, be working on a small emotional reserve and have little to draw on in times of stress.

Intellectually, the higher his standard the better chance a man has to readjust himself following an injury to his head. As a useful generalisation it can be taken that either physically, emotionally or intellectually, a man may be first, second or third class, and that his personality is conditioned by a complicated integration of his physical, emotional and intellectual endowments.

The importance of the type of man injured, in regard to prognosis of blunt injuries to the head, is well illustrated by the following personal experience and also by Russell Brain's analysis of 200 cases (see table on opposite page).

Some years before the war a pilot and his observer received severe injuries when their aeroplane crashed during an air display. Both the men were severely concussed; both of them were burned about the body and limbs, the pilot also receiving a compound fracture of the frontal bone with damage to the frontal lobe. Both of these men were unconscious for more than a week.

[1] Ascroft, P B "Traumatic Epilepsy after Gunshot Wounds of the Head" *Brit Med Jour*, 17th May 1941

As soon as the pilot began to recover, his first question was whether he would be able to fly again His mother, although deeply concerned about him, also stated that she felt it would be no good keeping him alive if he were not able to fly After a course of rehabilitation this man returned to the Air Force and later won the Victoria Cross in action over the battlefields of Tunisia.

The reaction of his observer was entirely different, as was that of his relatives Immediately his relatives realised that he was going to live, they expressed the opinion that he should be discharged from the Air Force. Immediately the boy himself became fully conscious and realised what had happened to him, he too said that flying was no job for him I was not able to follow the case up, but I believe that shortly after the injury he was discharged from the Air Force

ANALYSIS OF THE OCCUPATION OF 200 CASES[1]

Type of Accident	Occupation	Total	Neurotic	Percentage
Road	Professional and clerical	38	4	11
	Skilled artisans	27	5	[illegible]
	Light unskilled	14	6	[illegible]
	Heavy unskilled	10	7	37
	Unclassified	2	1	
Industrial	Professional and clerical	2		
	Skilled artisans	20	7	[illegible]
	Light unskilled	19	10	[illegible]
	Heavy unskilled	50	38	64

Nature of Injury—Occasionally a diffuse neuronal injury may be so severe and so much cerebral tissue destroyed that the patient is left permanently demented Fortunately this happening is rare. Loss of taste, diplopia, blindness or hemianopia deafness or impairment of locomotion will considerably aggravate the effects of damage resulting from the diffuse neuronal type of injury In minor injuries which could not possibly have led to permanent cerebral damage, resulting disability must be due largely to the influence of the other two factors that is to the type of man injured and to the conditions obtaining following accident

Following recovery from diffuse neuronal injuries of medium severity, actual physical damage to the brain can rarely be

[1] Brain, W Russell. *Proc Roy Soc Med.* 1942, 35, 304

demonstrated by clinical neurological examination or, in fact, by special examinations such as pneumencephalography. Even from electro-encephalography it is impossible to deduce how much of the brain tissue is functioning faultily or to localise the damaged area. This inability to be certain of the nature of the physical injury is one of the reasons why prognosis in the blunt type of injury is so largely a matter of conjecture and why we are compelled to depend for our judgments on previous experiences based on statistical surveys.

Circumstances obtaining after Injury.—Ideally, following skilled treatment in the acute stages of his head injury, a man is sent to a rehabilitation centre. There, amongst other things, he is encouraged, reassured, economic family difficulties inquired into and, if possible, satisfactorily settled. On the other hand, we have the man who is hustled out of a busy hospital as soon as he is conscious and sent home by ambulance to stay in bed until he feels able to get up and about In such instances the rehabilitation usually takes place in the kitchen over a hot fire, and under the supervision of his wife—in conditions which would soon make a healthy man feel ill.

Some of the injured have good jobs which they enjoy, but in some cases the opposite circumstances hold—a man may never have liked his work and for long has been anxious to leave it; injury gives him the opportunity to escape.

Disability is often prolonged by a desire for compensation or by the fear of instability. In some cases relatives, are naturally fussy and encourage a state of invalidism, whereas in others relatives are more stoical and encourage readjustment and early return to work.

RESULTS

In 1932 Russell made a statistical survey of the late symptoms following acute injuries to the head in a series of 200 cases. There were 16 deaths and, of the 184 cases which survived the injury, 141 were interviewed and examined at an average interval of six months after the accident; 55 of these had noticed no abnormal symptoms since discharge, while in the remaining 86 symptoms which were presumably due to the injury had been present since discharge. In his analysis of the common post-concussional symptoms, Russell[1] stated that it was considered advisable to exclude those cases in which there was any question of compensation outstanding; there were 14 such cases The remaining 72 cases were grouped according to the duration of loss of consciousness (see table on opposite page).

[1] Russell, W R "Cerebral Involvement in Head Injury" *Brain*, 1932, 55, 549

In 1942, Russell[1] carried out a further survey of 200 consecutive patients admitted to the Edinburgh Royal Infirmary The patients were interviewed eighteen months after their accident and the duration of any symptoms referable to the head injury were classified as follows —

Duration of Symptoms	Number of Cases
Under two months	80
Two to six months	11
Six to eighteen months	30
Over eighteen months	79

It will be seen from the table that in 40 per cent of cases there were no symptoms after two months, but in an equal number symptoms persisted for over eighteen months

Group A —Unconscious for less than one hour
Group B —Unconscious for from one to twenty four hours
Group C —Unconscious for more than twenty four hours

	Group			Totals
Degree of headache—	A	B	C	
Severe	1	7	5	13
Slight	11	12	5	28
None	8	8	15	31
Totals	20	27	25	72
Dizziness—				
Present	11	10	10	31
Absent	9	17	15	41
Totals	20	27	25	72
Impairment of memory—				
Severe	0	0	4	4
Slight	8	4	10	22
None	12	23	11	46
Totals	20	27	25	72
Nervousness—				
Present	7	7	11	25
Absent	13	20	14	47
Totals	20	27	25	72

[1] Russell W R "Medical Aspects of Head Injury" *Brit Med. Jour.*, 1942 2, 541

Cairns[1] (1942) stated: "I believe that almost every patient who makes a full recovery from concussion suffers at a certain stage of his recovery from headaches. In the mild head injuries the headaches may occur sporadically for some weeks and then disappear. In the severe cases the headaches may not come on at all if there is any serious degree of residual organic defect. The patient must have recovered a certain amount of mental clarity before he appreciates headaches. . . ."

In a series of 242 consecutive cases of injury to the head, Symonds and Russell[2] reported 5 deaths. Of the 237 survivors, 22 were invalided from the Services before they left hospital. The remaining 215 men were returned to duty, and of these 193 were followed up by questionnaire, which showed that another 11 per cent. broke down. This means that in the series of acute head injuries concerned, 80 per cent. of those who survived returned to duty and remained at their work for considerable periods. Moreover, the authors' statistics show that prolonged periods of rest in bed and prolonged convalescence give no better results than when patients are got on to their feet early and encouraged to return to duty.

In a follow-up of 132 Service cases from three to twenty-one months after injury, Botterell and Wilson[3] give the following analysis of the final results:—

Post-traumatic Amnesia	GROUP 1 *Nil.* Momentary	GROUP 2 *Momentary* 24 Hours	GROUP 3 1 to 7 Days	GROUP 4 Over 7 Days
TOTAL CASES	54	84	23	19
Unfit for duty because of other injuries	5	5	3	0
Transfers (other injuries)	1	10	4	1
Deaths	0	0	0	5
Remainder	48	69	16	13
NUMBER TO DUTY TOTAL	48	68	12	4
Category "A"	47	65	10	4
,, "B"	1	0	1	0
,, "C"	0	3	0	0
,, "D"	0	0	1	0
Unfit for duty because of head injury	0	1	4	9
Invalided later	2	0	1	0

[1] Cairns, H *Proc Roy Soc Med*, 1942, 35, 300
[2] Symonds, C P, and Russell, W R "Accidental Head Injuries Prognosis in Service Patients" *Lancet*, 2nd January 1943, 7-10
[3] Botterell, E H, and Wilson, K E "The Active Management (Non-operative) of Cranio-cerebral Injuries" Not yet in publication

In a series of 718 chronic cases admitted under the authors (Symonds and Russell) care from outside hospitals immediate discharge from the Services was effected in 31 per cent. Of those who returned to duty 21 per cent. were invalided later that is in the chronic group 52 per cent. finally had to be returned to civilian life The authors suggest that in the selected chronic group the large percentage of failures was due to the fact that many of the men showed a predisposition to mental disorder In support of this argument the authors state that in the flying personnel of the Royal Air Force, where the physical and mental background is good, the prognosis following head injury is excellent

In a series of 277 Service cases under the author s (Rowbotham) care the final results were as follows —

Military Cases—197

Returned to duty	58
Discharged to military convalescent depot	35
Transferred to E.M.S convalescent hospital	17
Invalided from Service	10
Downgraded	17
Transfers to other special centres	14
Deaths	8
Still remaining in hospital	11

Naval Cases—29

Returned to duty	20
Transferred to E.M.S convalescent hospital	5
Transfers to other special centres	4

R.A.F Cases—32

Returned to duty	16
Transferred to E.M.S convalescent hospital	5
Transfers to other special centres	9
Deaths	2

Women's Service Cases—19

Returned to duty	8
Transferred to E.M.S convalescent hospital	4
Transfers to other special centres	4
Invalided from Services	3

The above statements on the prognosis of head injuries in Service cases are so important that they merit further scrutiny The results were obtained under ideal conditions the patients themselves were young and healthy and, after having received skilled treatment in the acute stages, were formally rehabilitated and their final disposal decided by those medical officers under whose care they had been throughout their illness Moreover in Symonds and Russell s figures there were only 5 deaths in 242 cases, thus death rate being extremely low compared with figures taken from the larger voluntary hospitals where the rate is at least 15 per cent This must mean that the cases were diluted by minor types of injury

Mock,[1] in a survey of 6,462 consecutive injuries of the head over the years 1930 to 1940, found that the death rate between 1930 and 1935 was 35 per cent. and between 1935 and 1940 was 28 per cent.

Since the first edition of this book was published, the author has had opportunity to review some of the pronouncements made on the prognosis of closed head injuries resulting from road and industrial accidents and to analyse again the figures on which they were based.

Results of Road and Industrial Accidents.—One hundred patients who received blunt injuries to the head at least two years prior to inquiry have been interviewed; in all cases the injury was of the type which rendered the patient unconscious for at least twenty-four hours. Also, litigation had been finally settled in all cases where compensation was at issue. Seventy-five per cent. of these patients declared that it was six months before they were reasonably free from symptoms, and that it was two years before they had made their fullest recovery.

Full recovery from symptoms, of course, is not necessary before a patient can return to his former or to some kind of useful employment. It is to the possibility of early resettlement and early return to work that more attention must be devoted in the near future. With skilled medical treatment and correct advice, allied to new industrial litigation, far more men will be able to return to employment earlier than hitherto following injury. To generalise on prognosis from the favourable results of a small series of cases where all the conditions have been specially favourable is unfair to that large mass of injured people who have not had the advantage of highly skilled medical treatment and rehabilitation. The good results in any small series of cases should stimulate us to make more general those methods which have produced those good results.

Results of Injuries to Special Parts.—In an analysis of 400 consecutive patients suffering from the results of head injuries received not less than six months before, the findings regarding sense of smell and taste, vision and hearing were as follows:—

Vision.—Forty-six patients complained of varying degrees of impairment of vision. In 20 cases the loss of vision was merely a general deterioration of the order that necessitated their wearing glasses, whereas prior to the accident they had been able to see adequately without them; no doubt, in these cases old-standing refractive errors had been brought to notice by the accident. There were 6 cases of unilateral optic nerve injury in which the affected

[1] Mock, H E "The Management of Cranio-cerebral Trauma and Associated Injuries" *Surgical Clinics of North America*, 1942, 22, 989

eye was blind and there were 2 cases of injury of the optic chiasma in the above 8 cases the loss of vision will be permanent

Twenty four patients complained of diplopia. In 8 cases no squint was discernible, the disability being mild and improvement taking place, complete recovery almost certainly would occur in all of the 8 cases In 2 cases there was a third nerve palsy, in 2 a sixth nerve palsy and in 1 both the sixth and fourth nerves were involved, in 4 cases both the fourth and third nerves were affected Prognosis in these cases was doubtful, but I formed the opinion that many of them would probably recover In 5 cases there was displacement of the axis of the eye, in all these cases the diplopia will probably be permanent because they were also associated with injuries to the extra ocular muscles Pure displacement of the eye does not necessarily lead to diplopia as a good deal of compensation with fusion of the visual images can occur On the other hand, displacement of the eye associated with injury to the neuromuscular mechanism is an extremely grave combination Dislocation of the pulley of the superior oblique muscle is also apt to lead to permanent diplopia.

Deafness —There were 19 patients who complained of deafness, in 15 cases the deafness was unilateral and in 4 bilateral

In the 15 unilateral cases the deafness was complete in 7 and partial only in 8 In 1 case only was unilateral deafness of the nerve or internal ear type, and this was the result of a pyogenic meningitis which complicated a petromastoid injury

Of the 7 patients with complete unilateral deafness 2 had severe giddy attacks and 5 had persistent noises in the head Of the 8 patients with partial unilateral deafness, none were giddy, but 5 had persistent noises in the head

In 4 patients the deafness was bilateral, in 2 of these the deafness was severe, moderate in 1 and slight in 1 The deafness in the bilateral cases was of the nerve type in 1 instance only In none of the above 19 cases had there been any evidence of improvement after the first three months

Sense of Smell —There were 21 cases of loss of smell, in 8 there was loss of smell without loss of taste in 4 there was loss of smell with impairment of taste in 7 there was loss of smell and loss of taste In 2 cases patients complained of perversion of the sense of taste (parageusia)

Facial Paralysis —It is of interest to note that in none of the above 400 cases was there an instance of residual facial paralysis

Long-term Effects.—Even though resettlement and correct legislation and treatment will expedite a man's return to work and thereby improve his economic situation injuries to the head are none the less serious in their long-distant effect

In my follow-up of patients at least five years after their accidents, I often heard a wife state that no money could put right what the accident had done to her husband: that "this is not the man I married" and that "the accident had completely changed him." Often a wife has had to take over the family responsibilities while her husband sits back introspectively and has to be humoured, encouraged and guided. Severe injuries to the head, in fact, nearly always produce long-standing and far-reaching emotional and dispositional changes

We readily accept that injury can cause a hemiplegia because the concomitant structural cerebral change is easy to demonstrate and because locomotion has a fairly precise representation in the brain. On the other hand, many people doubt that physical damage to cerebral tissue can produce alterations in disposition and deterioration in concentration, because the representation of these thought processes in the brain is not known; therefore, they are commonly attributed purely to psychological influences or to psychoneurotic tendencies. This, I believe, is a mistake. It was Socrates who first suggested that thinking stands in the same relationship to the brain as does a musician to his instrument; in other words, thinking arises outside the brain in the universe or in the stream of life, the brain acting merely as a receiving set. Even if this hypothesis is true, the quality of thinking must be affected by the quality of the instrument which produces it in the form in which we can appreciate it

Finally, I should like to say that our future object should be to prevent rather than to cure head injuries

CHAPTER XIII

THE MECHANISM OF BIRTH INJURIES[1]

DURING labour, as the fœtal head leaves or attempts to leave the uterine cavity, it is subjected to those stresses and strains which result from the forces of natural expulsion and of obstetrical manipulation acting against the resistances of the maternal passages. Uterine contractions, of course, are the forces by which the majority of children are born, but these are usually augmented by the voluntary contraction of the abdominal muscles. Forceps extraction, traction on the legs in breech presentations, or pressure by the accoucheur on the abdomen can aid or completely replace the forces of propulsion. Resistance to downward progress of the fœtal head may occur at a Bandl's ring, at a rigid cervix, in the pelvic passages or against the perineum.

Let us consider in more detail the various factors concerned. The fœtal head is a complex body consisting of a loosely constructed dome set on a relatively flat rigid base. Each bone of the calvarium at this stage of calcification is extremely plastic and can easily be made to alter its shape without breaking. Moreover, each bone is attached to the next by a fibrous membrane which allows it to slide over or to pivot freely on its neighbour. Thus the fœtal head is so constructed that it can be deformed both locally and generally to a considerable degree without injury being inflicted on it as a whole or on any of its component parts. As will be seen later, it is this ready malleability of the head that accounts for the type of cerebral damage which so commonly occurs from birth trauma. Although none of the intracranial contents can be compressed into a smaller volume by force of the magnitude concerned, the cubic capacity of the head can be lessened to an appreciable extent by displacement of venous blood or of cerebrospinal fluid into the veins of the neck and into the spinal theca respectively. By this mechanism the cerebral tissue can, within certain limits, be protected from excessive generalised intracranial pressure when the head is being forced through the pelvic passages.

[1] Beneke, *Münch Med Wochschr.*, 1910, 57, 41
[2] Holland, E., *Jour Obst and Gynæc of Brit Empire*, 19[illegible], 29, 17

The dura mater is a most important structure in preventing and determining the nature of injury inflicted at birth (Fig. 253)

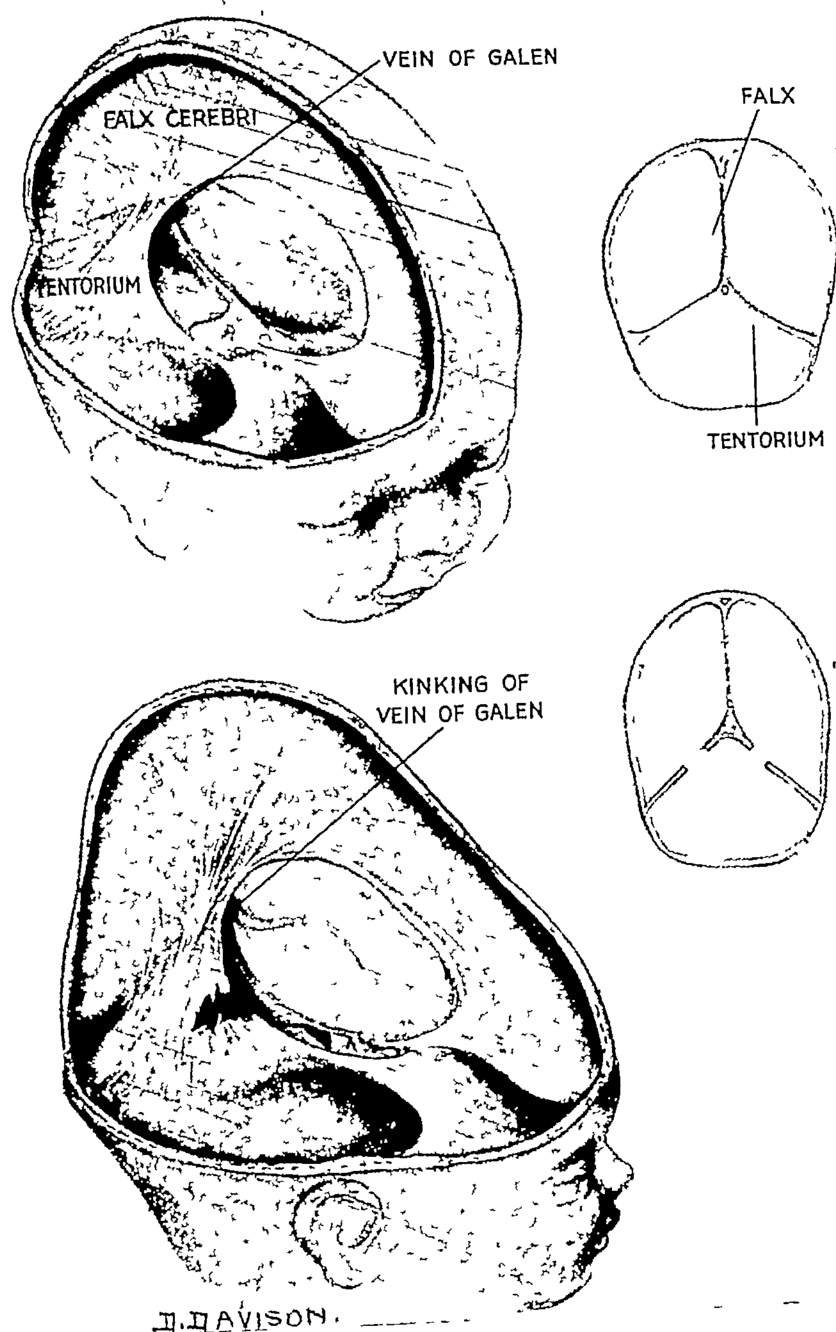


Fig 253

Stretching of the falx and tearing of the tentorium in moulding

From the obstetrical point of view, the inner layer of the dura mater may be regarded as consisting merely of flattened cells lining the inner surface of the outer fibrous layer. These cells secrete a lubricating fluid which allows the arachnoid mater to

slide freely across the dural interface without tearing and thus protects the leptomeninges from injury during the processes of moulding of the fœtal head. Injury to the cells of the inner layer, on the other hand, may lead to excessive secretion and to the formation of a subdural hygroma possibly of sufficient size to compress the brain. Occasionally, as in adults, bleeding occurs into the subdural space.

It is, however, with the falx cerebri and the tentorium, the two large folds of the fibrous layer, that we are primarily concerned. The convex base of the falx cerebri which measures at least 1 in. across is firmly attached to the vault of the skull from the root of the nose anteriorly to the internal occipital protuberance posteriorly. From the lateral boundaries of its superior attachment two layers of membrane converge to enclose the sagittal sinus and join to form the falx proper. Inferiorly the falx from before backwards is attached to the crista galli; then it arches over the corpus callosum and finally blends with the tentorium itself, two layers here separating to enclose the straight sinus.

The tentorium lies roughly in a transverse plane at right angles to the falx. Posteriorly, it is firmly attached to the internal occipital protuberance and on each side to the lateral wall of the skull. Anterolaterally it is attached to the petrous ridge of the petrous bone and by special ligaments anteriorly to the clinoid processes. Its anterior border is concave and with the dorsum sellæ encloses the opening occupied by the midbrain. It forms the roof of the posterior fossa and it slopes upward from its periphery to the line along which it blends with the falx cerebri.

The nature of the venous drainage of the brain is also a factor in determining the type of damage that follows birth injuries. The cerebral veins drain into the dural sinuses and the connecting veins are short and thin walled, rupturing easily when they are put on the stretch. The veins of the choroid plexus join the veins of the corpus striatum to form the small veins of Galen; the small veins of Galen join together beneath the splenium of the corpus callosum to form the large vein of Galen and this joins the inferior sagittal sinus to form the straight sinus. It is this junction which is so important in determining the type of injury which occurs at birth.

At the start of labour the fœtus is in an enclosed cavity surrounded by fluid, a system governed by the physics of hydrostatics. So long as the membranes remain intact and the liquor amnii is retained, rises in intra-uterine pressure resulting from uterine contractions are distributed uniformly and the fœtus is subjected to compressing forces only, shearing forces being

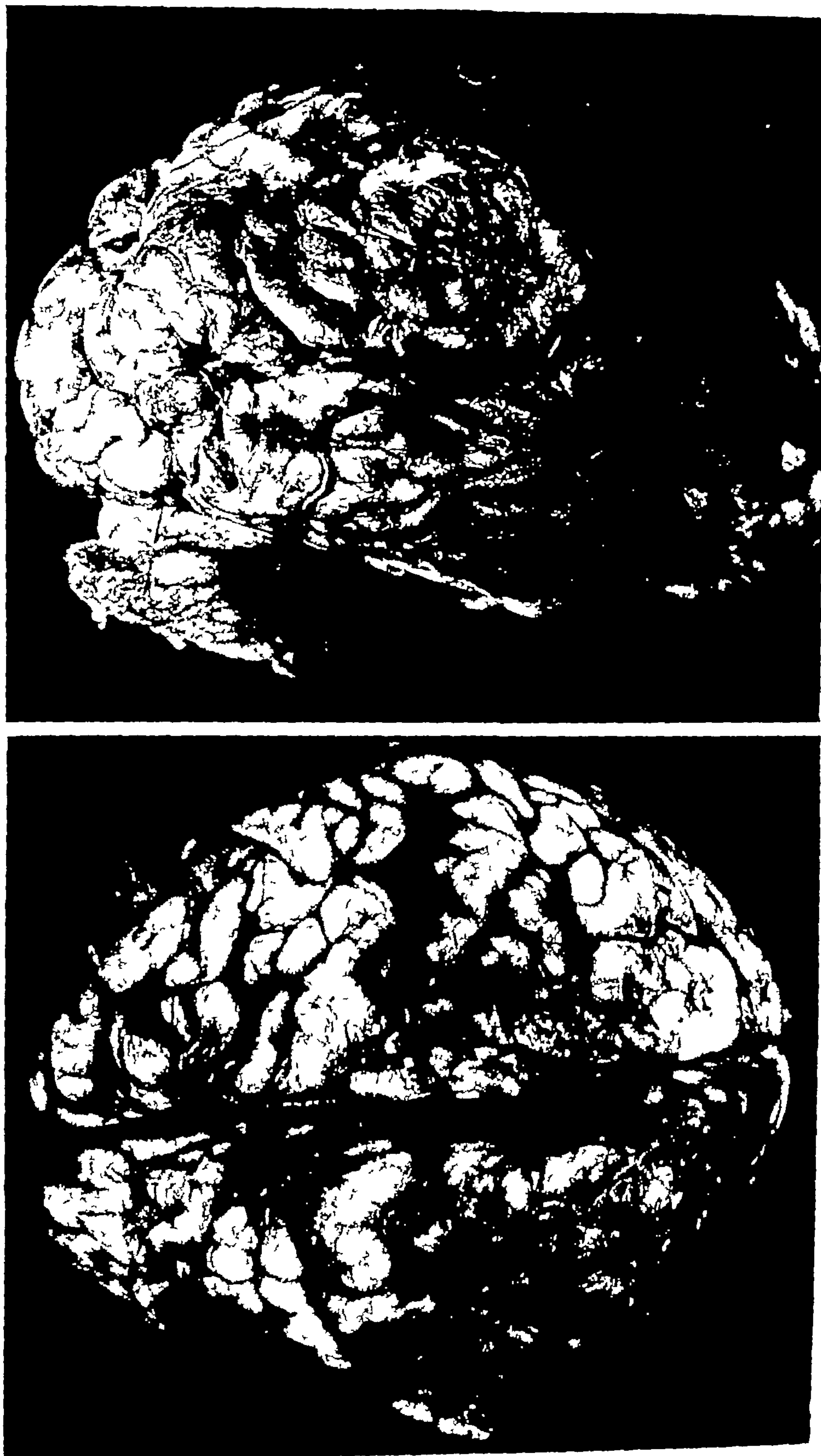


FIG 254
A convexity hæmorrhage in a new born child

absent. Compressing forces of the magnitude usually involved do little harm as they tend merely to push the fœtus into a smaller volume and not out of shape In prolonged labour however, even with intact membranes, and still more when there is premature rupture of the membranes and loss of liquor, fœtal distress may be caused either by direct pressure on the fœtus or as is more probable, by compression of the placental site Death in fact, *can occur in the first stage of labour when no other cause* can be found except strong and prolonged uterine contractions At autopsy cerebral hæmorrhages have been demonstrated in children born by Cæsarean section where no actual delivery trauma could have occurred In those cases where the membranes rupture prematurely little harm is usually done since the loss of the protecting liquor is minimised by the presenting part filling the lower part of the uterus and closing off the cervix from the body Relaxation and retraction of the cervix have been proved to be the result of an active muscular action and it is probable that dilatation by the cone or wedge-like action of membranes or of presenting part plays a small part in the process of the opening of the os In cases of primary inertia however no co-ordination of muscular activity is present and the cervix may remain undilated for long periods This lead to attempted moulding of the head and the production of shearing forces which may lead to cerebral damage

Normally, as the os dilates, the membranes rupture and the presenting part of the fœtus which for our purposes we will regard as the head, becomes engaged in the cervical ring The scalp projecting through the cervix becomes congested and swollen because the veins at the periphery become compressed and obstructed before the arteries The head is then pushed onwards through the pelvic passages by the thrust of the baby's body acting through the spinal column and occipital condyles The physics are such that the head rotates until its greatest diameter engages the greatest diameter of the pelvic passages However, the shape of the head in any given plane may not, and probably in fact often does not, exactly correspond with the shape of the pelvic opening in which the head may be lying Also the head soon becomes engaged over a considerable length in a longitudinal axis which means that however perfectly the head fits the pelvic passages, some degree of moulding must occur to allow the head to pass. The amount of moulding necessary depends on how well the head fits the pelvic passages In faulty presentation in deformity of the pelvis when there are rigid soft parts and when the head is unduly large, moulding is always considerable and often excessive

adults the head is subjected to sudden severe violences, the fœtal head is subjected to prolonged forces of compression and distortion.

Cerebral damage may occur in precipitate labour either by excessive compression, too rapid moulding or by falling after the child is born.

Prematurity because of pathological fragility of the cerebral tissues seriously predisposes to injury and explains why severe damage may occur in normal labour. It is because of the liability to produce premature birth and cerebral injury that syphilis so often leads to a stillborn child. Also diseases which cause increase in the clotting time of blood seriously aggravate birth injuries, as these are so particularly liable to be of the hæmorrhagic type.

The Scalp.—The caput succedaneum is a normal phenomenon of birth and is due to venous congestion and œdema resulting from ring compression of the scalp. Only when it becomes excessively large and an obstruction to labour can it be regarded as pathological; in these cases it will usually be found that blood has extravasated into the tissues. Bruising of the scalp is common in difficult labours, but laceration in labours unaided by instruments is exceedingly rare.

The Skull.—The bones of the fœtal head are so plastic and glide so freely over each other that a great deal of distortion is necessary before they are bent beyond the limits of their elasticity. It is by bending that fractures or indentations of the bones occur.

Indentations, either rounded or pointed, can occur in unaided or in instrumentally aided labour. In unaided labour indentation may occur, though very rarely, by pressure on the sacral promontory or against some abnormal projection. Indentations which result from squeezing against the sacral promontory are nearly always situated in the parietal area just above the ear; in other words, they occur in the widest diameter of the head They are not usually associated with fractures, although the inner table itself may be broken.

With instrumental injuries, indentations are often more pointed and more extensive than the usual medium-sized saucer depressions of unaided birth; the more pointed a depression, the more likely is an associated fracture to occur. The tendency is for a saucer-shaped depression to be elevated by natural processes as the brain develops and increases in size. Occasionally, as the head is pulled out by forceps, an indentation may occur against the promontory (Fig. 256).

Fractures are nearly always the result of instrumental labour and usually occur where the blades are applied. Occasionally

fracture is confined to the inner table They are, however, rare except in very difficult operative deliveries

In malformed pelves the head may undergo such excessive moulding that permanent distortion of the head results Whether such a condition is the result of bending of the bones beyond their limits of elasticity is not known Very probably it is the result of rupture of those dural struts which are so placed to maintain the normal shape of the head The classical condition known as cephalhæmatoma consists of a hæmorrhage beneath the pericranium. It is therefore restricted to the confines of the bone in relationship to which it forms It may or may not be associated with a fracture of the underlying bone



FIG. 206
Indentation at the age of two months.

The Brain.—There has long been controversy on the type of injury which usually results from birth trauma. From the literature on this subject one gathers the impression that tears of the dura with resulting hæmorrhages are the main if not the only form of damage Hæmorrhages of course cannot *per se* cause neural dysfunction save by generalised anoxia. They can affect the brain directly only by compression in virtue of their mass or by hydrocephalus the result of blockage of the cerebrospinal fluid pathways with red blood corpuscles Infarction of course can occur as a result of rupture of the vessel concerned

Anoxia—Anoxia probably is the main cause of the damage which is inflicted on the brain during labour In the compressions and distortions to which the head is subjected blood vessels must be stretched kinked and flattened with resulting ischæmia. Venous congestion is a form of faulty circulation and leads to

faulty oxygen metabolism Asphyxia is but a form of anoxia. Momentary but complete deprivation of oxygen to the cortex leads to permanent damage or to death of the neurones concerned. The medullary cells are a little less vulnerable to oxygen lack, otherwise more children would die as the result of labour Strangulation of the cord is another cause of anoxia In breech deliveries the too forcible manual compression of the abdomen and chest will force blood up into the brain and, apart from obstructing cerebral circulation, may result in rupture of cerebral vessels and hæmorrhage. Anoxia not only leads to acute manifestation of neural dysfunction but also to all kinds of permanent paralyses and mental deficiencies. According to Dr J. Linton Snaith [1] almost complete atelectasis of the lungs is a common finding at post-mortem in those babies which are born in a state of white asphyxia and which are slow to breathe, cry and suck and prone to repeated attacks of blue asphyxia. In other words, true asphyxia is the underlying cause of many of those cerebral conditions seen in children shortly after birth which are thought to be due to direct trauma to the head

Hæmorrhages.—Subarachnoid bleeding in birth injuries is almost invariably present, and is, in fact, common in children who show no evidence of injury to their heads

Convexity hæmorrhages may be extradural, subdural or subarachnoid. Extradural hæmorrhages are extremely uncommon Subdural and subarachnoid hæmorrhages usually result from the rupture of the superior cerebral veins. Subdural hæmorrhages are not rare but are usually associated with subarachnoid bleeding. In subdural hæmorrhages, blood, apart from seeping diffusely over the subdural space, may collect in clots of considerable size over the convexity of the brain. The usual position for such subdural hæmatomata is high in the parietal region either on one or both sides. They can, however, occur over any part of the brain.

Basal hæmorrhages result, as a rule, from rupture of the veins of Galen, of the straight sinus or of the tributaries of the straight sinus, and are associated with tentorial tears. In these cases clots of blood are often seen above and below the tentorium and in the cisterna ambiens. Diffuse subarachnoid bleeding is, however, the dominant feature and blood collects in the pools of the cerebral and cerebellar cisterns The volume of blood extravasated may be such that the intracranial pressure is raised and the brain compressed. Pressure on [illegible] bstruction of the veins of Galen causes congestion in the [illegible] of the brain drained on each side by the veins of the co[illegible] and of the choroi[illegible] plexus.

[1] Personal cc

Intracerebral hæmorrhages are rarely massive being usually of the diffusely scattered petechial type They occur either as the result of torn vessels or from diapedesis The petechial hæmorrhages in themselves rarely do harm, save when they occur in an important pathway, for example about the internal capsule They may increase anoxia by increasing associated cerebral œdema.

Ventricular hæmorrhages are usually severe and it is believed they are almost invariably fatal They result either from rupture of massive intracerebral hæmorrhages into their cavities or from tearing of the choroid plexus

When anything goes wrong with the gross physical and physiological workings of the brain as a result of trauma a vicious circle develops A child with cerebral injury breathes badly this causes asphyxia, and the resulting anoxia aggravates those conditions which were originally caused by oxygen lack Ischæmia leads to convulsive seizures and these, in turn, lead to further cerebral venous congestion and to circulatory embarrassment As in adults, metabolic crises occur as probably do other conditions of which fat embolism is an example

Sequels.[1-3]—Little's disease or cerebral diplegia is one of the best known ailments of childhood Clinically the picture is clear cut, the paralysis being more marked in the legs than in the arms It is doubtful whether the condition results from trauma although it certainly may occur as a result of traumatic thrombosis of the superior longitudinal sinus Probably the essential pathology is an arrest or fault in development of the pyramidal pathway The condition is associated with a diffuse cortical atrophy and pneumencephalography reveals an enlargement of the body of the lateral ventricle Little's disease is commonly mistaken for the condition of double hemiplegia which almost always results from trauma. In double hemiplegia the paralysis is more marked in the arms than the legs Unilateral hemiplegia in many cases is the result of trauma. Porencephaly may be due to developmental faults or to trauma

Hydrocephalus—Hydrocephalus which develops within the first few weeks of life and which may not have been evident at birth, is most commonly due to arrest of or fault in development of the leptomeninges More rarely it is due to non patency of the aqueduct of Sylvius (Fig 257)

[1] Penrose L. S. "Birth Injury as a Cause of Mental Defect" Paper to be published in *Journal of Mental Science.*

[2] Norman, R. M "Cerebral Diplegia following Birth Injury" *Brit J Med Chir J* 1948, 65, No. 231

[3] Norman, R. M *Atrophic Sclerosis of the Cerebral Cortex associated with Birth Injury* *Arch. Dis. Child.*, 1944 19, No. 99

Trauma may lead to hydrocephalus either by causing stricture of the aqueduct of Sylvius or by obliteration of the cerebrospinal fluid pathways, the result of traumatic adhesions. A chronic subdural hæmatoma occasionally leads to extensive hydrocephalus.

Epilepsy.—In epilepsy there is first a predisposition which is inherited or produced either by disease or trauma. The actual

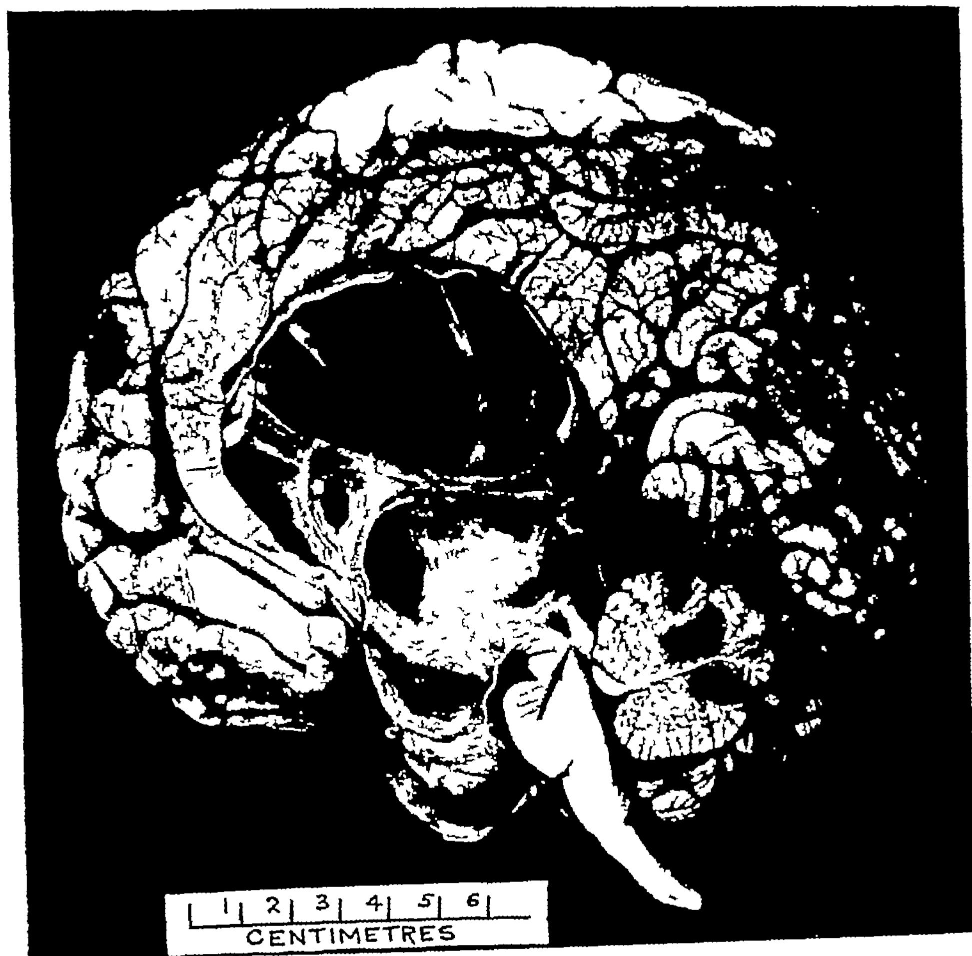


FIG 257
A stricture of the aqueduct of Sylvius leading to hydrocephalus

precipitation of an attack of epilepsy is caused, as a rule, by some minute metabolic change which sets the abnormal neuro-pathological mechanism into action. Probably, therefore, birth injuries of all degrees, types and dimensions are important etiological factors in the production of epilepsy. Certainly diffuse cortical atrophy which so often follows trauma can commonly be demonstrated by pneumencephalography in child epileptics.

DIAGNOSIS

A child that receives a severe injury to its head during labour is born with a bad colour; blueness or ... or is, in fact, the first

evidence of cerebral damage Faulty breathing is the second most obvious sign of cerebral trauma Normally a child draws a breath as soon as its chest is free and soon begins to cry When injured about the head resuscitation is difficult respiration may be absent, gasping or slow and irregular in rhythm The normal cry of a baby consists of a series of musical notes, in the head injured the cry may be meningeal in character the note being shrill and monotonous Thirdly, the anterior fontanelle instead of being flat may be bulging tense and non pulsatile

The general neurological picture is no different from that in adults described in Chapter III, save that certain normal differences in movement and reflex activity have to be taken into account because of imperfect myelinisation of the pyramidal pathways, in particular, the plantar reflex is extensor and grasp reflexes in the hands are present.

Abnormal posture is a common sign. The legs may be rigid in extension and the arms rigid in flexion, as seen in decerebrate rigidity, a state indicative of brain stem injury or compression In serious cases the position is that of opisthotonos indicative of a combination of meningeal irritation and pressure in the posterior fossa

One limb, one side or both sides of the body may be paralysed The eyes may move inco-ordinately or there may be a fixed strabismus The pupils may be of different size and [illegible] small there is a fixed dilated pupil on one or both sides Convulsive seizures are a common result of cerebral injury One side of the body only or one limb only may twitch Unilateral signs are particularly helpful as they are of localising value Unilateral paralyses and unilateral convulsive seizures point to a lesion in the opposite cerebral cortex Alternatively a fixed dilated pupil is indicative of a tentorial herniation on the same side Rise in intracranial pressure can be measured by spinal puncture and manometry, and by puncture the presence and severity of subarachnoid hæmorrhages can be established

In severe injuries the child's condition usually rapidly deteriorates and death ensues within a few hours In the less severe degrees of injury the child may make what appears to be a satisfactory recovery from the immediate cerebral crisis later to sink into the state of pallid or blue asphyxia or of status epilepticus *In the mild type of injury there may* be no frank signs referable to the brain, save that resuscitation is slow and suckling awkward and weak, on the other hand, sufficient damage may have been done to the brain tissue to lead to serious sequels. In injuries of medium severity *the whole of* the brain seems to be affected, which, in fact it is In most of the cases the condition appears

to be beyond the aid of surgical measures. None the less, detailed and careful examinations are worth while, since lives can be saved by suitable surgical procedure.

The problem of diagnosis is partly that of localisation and partly that of determination of the nature of the pathology of the lesion. A rise in intracranial pressure, as measured by manometry or shown by bulging of the anterior fontanelle not due to straining, is indicative of intracranial bleeding. The presence of blood in the cerebrospinal fluid, it must be remembered, does not prove that there is not a clot over the cortex. A latent interval or slow deterioration must always be regarded as meaning hæmorrhage.

The localisation of a lesion depends on localising neurological signs, such as unilateral paralysis or convulsion or differences in the reflexes on the two sides. Occasionally a superficial lesion, such as a bruise or a cephalhæmatoma, will point to the site of the underlying and deeper blood clot

From the therapeutic point of view the main object of detailed examination is to detect localised bleeding which is seriously compressing the brain and which is in a position suitable for surgical treatment. Such hæmorrhages are virtually confined to the subdural type situated over the convexity

The signs of a convexity subdural hæmorrhage are as follows —

(*a*) A rise in intracranial pressure demonstrated either by lumbar puncture and manometry or shown by bulging and loss of pulsation of the fontanelle

(*b*) Localising signs, such as unilateral weakness or paralysis and convulsions

(*c*) There may or may not be a fixed dilated pupil or signs of superficial damage on the head.

(*d*) Possibly the most important sign of all of a hæmorrhage amenable to surgical treatment is a latent interval or slow deterioration of the child's cerebral condition.

TREATMENT [1]

Sound and careful obstetrical judgment may prevent the occurrence of a serious head injury at birth by avoiding prolonged labour or the necessity for a high forceps delivery. Great skill and experience is needed, for example, in the correct use of Cæsarean section.

It has been proved that there is a relative deficiency of prothrombin in fœtal blood which is greater in premature infants. The deficiency of prothrombin allows continued oozing from the

[1] Cushing, H *Amer Jour Med Science*, 1905, 130, 563

rupture of minute vessels which normally would readily seal and thrombose. Increasing use is therefore being made of a synthetic vitamin "K" in the prophylaxis of cerebral trauma. The vitamin may be given orally or intramuscularly to the new born child or, better still, to the mother during labour In the Maternity Unit at the Newcastle General Hospital the following routine has been adopted, and it *is claimed that* it has produced a reduction in the number of 'cerebral' babies One cubic centimetre of vitamin "K" is given intramuscularly every six hours during the first stages of labour to all primipara. The same therapy is carried out in multipara in which cases a pelvic abnormality exists and when, in fact, labour has been difficult or prolonged One cubic centimetre of the vitamin is given *immediately* to a new born child following a difficult labour or whenever there is the slightest suspicion of cerebral damage

Any anæsthetic or hypnotic drugs given to the mother during labour will affect the infant which may be born anæsthetised or drugged, and either condition is serious when the respiratory centre is already embarrassed by trauma Whenever a general anæsthetic is given great care must be taken to ensure a free airway and adequate oxygenation of the mother [illegible] a mild degree of cyanosis in the mother may have serious consequences in the fœtal brain The use of local anæsthetic for Cæsarean section and pudendal nerve block for forceps and breech deliveries minimise the risk to the fœtus Whenever a child is born with a cerebral injury treatment must be governed by [illegible] principles In particular, the whole of the child's metabolic processes have to be taken into consideration (the reader [illegible] to Chapter IV) Respiration must be started as soon as possible the child must be kept warm but not overheated and a correct fluid balance must be maintained Continuous oxygen should be given in cases of cyanosis

In those cases where there is raised intracranial pressure and cerebral embarrassment not due to an obvious local blood clot the pressure should be lowered by lumbar drainage to 50 mm of cerebrospinal fluid

Few doctors are prepared to submit a newly born child to any kind of major operation This of course is a sound basic principle On the other hand, a newly born child stands operative procedure reasonably well given that the blood loss is kept down to a minimum and that the manipulations are gentle and expeditious. This important point has been very clearly demonstrated by Professor Lambert Rogers[1] Therefore when a

[1] Rogers, L. *Brit Jour Surg.*, 1941 29, 3 "[illegible] Infant Twenty Minutes Old"

new-born child's life is in danger and a clot over the convexity of the brain has been diagnosed, the clot should be removed surgically. This is best done by making suitable burr holes, washing out the clot and draining the cavity. When the clot is solid and cannot be removed through local burr holes, a formal bone flap should be turned down. This is not a difficult procedure because at this age the bone itself can be quite easily cut with stout scissors.



(a)



(b)

FIG 258

A cephalhæmatoma

(a) Before operation (b) After aspiration

A cephalhæmatoma should be aspirated by means of a needle and syringe with strict aseptic precautions. It is unnecessary to incise and drain a hæmatoma particularly as this involves an added danger of infection. A warning is necessary here. Occasionally a congenital maldevelopment consisting of a defect in the calvarium and an underlying and bulging cavernous angioma closely simulates a traumatic cephalhæmatoma. If an angioma is incised the consequences are serious. Usually an angioma pulsates, but may not do so obviously, and there are usually no angiomatous markings in the skin. Therefore, whenever the pathology of a superficial swelling of the head is in doubt it is best to explore it with a fine-bored needle

CHAPTER XIV

THE RESIDUAL ILLNESS

WHATEVER may be the etymology of the word psyche it has now come to mean the mind It is impossible satisfactorily to describe or define it but none the less we all think we know what is meant by it Whether the mind originates within the body or outside of it is not known what we do know is that mental activities are paralleled or accompanied by physical activities of the brain There is no doubt that *physical changes in the neurones of the brain can* produce mental disturbances, and closed injuries of the head are especially apt to injure the mind since the physics are such that the whole brain is of necessity damaged at the time of accident A difficult problem is whether and how the mind can be injured by damage to parts of the body other than the [illegible] In considering this problem it must be [illegible] scalp, bone and dura from which [illegible] arise are not part of the brain in [illegible] this respect are comparable with [illegible]

There are two aspects of th[illegible]

(*a*) The physical messages th[illegible] which proceed to the [illegible]

(*b*) The contemplation of the [illegible] quences

On physical grounds it can [illegible] of injury the brain can become [illegible] arising from the periphery that [illegible] interfered with For example, a ba[illegible] they reach the conscious level, ma[illegible] physical activity of the brain at a [illegible]

Once a painful impulse has reached the [illegible] easy to understand how a persistently disagreeable sensation can distort the way of thinking or preoccupy the mind a[illegible] Can *a disagreeable sensation after it has reached consciousness* persist and develop without further reinforcement by repeated stimuli from the original physical focus of disturbance? We believe that it can, and that such a mechanism may well underlie some of the difficulties that are met with in the post concussional

syndrome. The contemplation of damage done to the body, and of its consequences may obviously lead to anxiety, and to a change in outlook.

We must now consider the so-called psychological injury which is the most difficult aspect of the problem. A man may witness an event which endangers the life or well-being of (*a*) himself, (*b*) someone he loves or (*c*) a stranger.

In such cases, however, it must be realised that the picture of the frightening scenes has passed to the man's brain by his visual pathways, and thus at one stage a physical mechanism was active and essential for the infliction of the injury Is it possible to create a thought within the mind so frightening that a lasting injury is thereby inflicted on the mind? Certainly an imagined wrong can disturb a man's outlook and make him unhappy. Such injuries are the only true variety of psychological injury

Let us next consider the meaning of the term psychoneurosis. It means a disturbance of the mind without loss of insight which leads to disturbance of behaviour, but which is not caused by permanent neuronal change in the brain. There may, of course, be considerable pathological changes in the brain, but the underlying cause is not a neuronal-anatomical one, as it is, for example, in a hemiplegia

In many cases of so-called psychoneurosis we feel that the patient's complaints are genuine, and are convinced that the patient is profoundly unhappy Indeed the essential nature of the illness may be regarded as pathological unhappiness. Such patients are often voluble, demanding, exacting and vindictive. They have a genius for causing trouble amongst their relatives, between doctors, between doctors and relatives and between a doctor and his nursing staff Many of us have suffered from their repeated telephone calls made at inconvenient hours. We know with what skill a psychoneurotic can tell a slightly different story to those in charge of his case designed to engender emotional unrest and conflict. Nevertheless whatever may be his stratagems, the patient remains profoundly unhappy, and little seems to help him. His illness comes from within himself and is not conditioned by external circumstances: it certainly cannot be cured by simple social adjustments.

On the other hand there is a variety of illness which, in some way or other, may advantage the patient. He might evade the responsibilities that he feels too great for him, or a job that he does not like; he might gain attention from someone whom he believes is neglecting him or be enabled merely to indulge a taste for laziness. Often, and on the demand of the patient, this illness is given a technical name to protect the patient's pride or self-

esteem That anxiety can eventually lead to a genuine physical illness is beyond doubt, as evidenced for example, by duodenal ulceration. But where is the line to be drawn between illness and a defect in personality or character? At what point does a patient become ill instead of lazy? What is going to be our therapeutic attitude? We know that in the recent past there has been a tendency for some doctors to give a patient what the patient thinks is good for him, for example if a man feel ill because of having to go into danger, the obvious remedy is to relieve him of the necessity of going into danger Possibly in view of present social conditions and because of our English way of thinking this might be the best way out of a difficult problem

To every illness there is of course a mental reaction of some kind In many cases the response is reasonable By reasonable we mean that the degree and nature of the reaction is very much what our own would be if we had received the same kind of injury, and if conditions were such that we had nothing to gain by remaining ill and were fond enough of our work to wish to return to it. These reasonable reactions should be regarded as psychological rather than psychiatrical problems [illegible] the duty of every doctor whatever illness he may [illegible] these reactions, to see the problem [illegible] obsessed by conventional therapeutic [illegible] that patients commonly ascribe [illegible] particular a headache is often attributed [illegible] the head when its origin is in fact [illegible] The right information will usually [illegible] careful, repeated and kindly enquiries When [illegible] is thought to be unreasonable the problem becomes [illegible] psychiatric and should be referred to a psychiatrist

With these general considerations [illegible] examine the post concussional compl[illegible] instructive to present the problem [illegible] describe the various phases in the order in which knowledge and experience were gained About twenty [illegible] interviewing patients who had received injuries [illegible] discovered what few signs such patients had to show for their injuries It was truly remarkable the speed and apparent thoroughness of the physical recovery that was made by patients who, only a month or so previously had been observed in a state of deep unconsciousness as a result of accidents on the road or in industry The complaints of these patients were largely of headaches and dizziness and similar subjective sensations With relatively few exceptions, detailed examination and special investigations failed to demonstrate any satisfactory physical

basis for the patients' symptoms Without a demonstrable physical cause, it is extremely difficult to determine how much a man is disabled by headaches or by dizziness because one's judgment on these matters must depend on the man's own words as to how much his illness affects his behaviour and on the statement of his relatives, and such indirect measurements must necessarily lack precision The problem, however, of the residual illness was found to be more complex than the mere assessment of severity and incapacitating effects of symptoms such as headaches, because it was obvious that the symptoms of which the patients particularly complained could not satisfactorily account for so much apparent inability to return to work and for so much anxiety in the relatives concerned Having had a thorough introduction to the post-concussional problem, I proceeded to study problems of litigation Fortunately the conditions of this work are such that careful enquiry and detailed records are necessary and I now speak from the experience of one thousand cases. The experience of litigation gave me a deeper insight into the mental aspects of head injury than I had gained in the Out-Patient Departments of my hospitals.

In most instances of litigation I formed the opinion that my patients were well aware of the true consequences of the injury they had received, both for themselves and for their families. Nearly all the patients, even when they had received a severe injury, were well aware of the significance of even the most intricate financial arguments, indeed, if a mistake was made to their detriment they quickly noticed it and wanted it corrected. On the other hand patients often were most stupid about and resistant to advice which would, if followed, in the long run have been beneficial to them even in the strictly financial sense. For example, a man would stick out for a few hundred pounds and, by the time he got it, he must have lost two or three times as much in wages or in other ways. Often when pressed to accept a settlement which would have been best in a long-term sense, a patient became antagonistic and would show his resentment by unreasonable suspicion. In cases where it was obvious that the counsellor could have nothing but the interest of his patients at heart he was often told that he would talk differently if the accident had happened to him, implying that his advice was not, after all, totally disinterested, which in fact it was.

Even after the closest scrutiny and fortified with the awareness of the possibility, I rarely came to the conclusion that a man was a frank malingerer This conclusion I know cannot be proved because the English way of thought precludes any secret investigations or spying into the way an injured man behaves in his private

life. On the other hand I was often convinced that a man was exaggerating his symptoms in order to gain his end, and the end was as often emotional as monetary. Indeed it was often obvious that a man was fully enjoying the fuss and sympathy his family was giving him and had no intention of giving up easily what to him was a great and sometimes unprecedented luxury. Whilst in legal conference, and when a barrister was probing the weakness of his case, it was rare that a head injured litigant fell down before even the fiercest barrage of questions. For example, a man rarely had too little or too much to say; he rarely got angry or flushed when pressed and would often gain a point by just the correct gesture or the correct facial expression. In the witness box of the open court the head injured litigant is notoriously an excellent witness. That a man is a good witness, it may be claimed, indicates the genuineness of his plea. On the other hand I believe it also implies that his intelligence has not been seriously impaired.

The more one studies litigation the more one asks oneself whether or not a litigant is gro[illegible] and whether he will not quickly [illegible] recompensed. What does a man [illegible] sum settlement? Does a man [illegible] severe closed head injury and [illegible] disability if any?

I gradually came to appr[illegible] needed on the problem of the [illegible] therefore decided to interview [illegible] the patient, and to follow the [illegible] how they reacted there. It w[illegible] got the best insight into the re[illegible] With [illegible] wives and mothers stated fr[illegible] husbands or sons had received [illegible] *recompensed them for the way they* [illegible] result of the changes in personality [illegible] chiefly in the home that the effects of injury were felt [illegible] of negativism, of emotional lability, of unhappiness [illegible] temper of intellectual deterioration and of laziness.

In fifty instances detailed enquiries were made into what happened to the moneys of lump sum settlement. On the information given in this enquiry I can state that even adequate compensation does not cure a traumatic cerebral illness. On the other hand lump sum settlement in the early convalescent stages often expedites the return to useful employment because a man now feels that it is up to him to readjust himself to the best of his curtailed ability. In other words, compensation clears up a

patient's exaggeration but not his medical disability To solicitors it therefore can be said that the "golden ointment" is not curative, but useful.

In four instances the moneys of a lump sum settlement were rapidly squandered in lavish generosity to relatives and friends and in the expenses of parties, racing and alcohol. In no instance were there any regrets by the man himself regarding the way the money had been spent

In six instances attempts had been made to establish little businesses with something like success in two only. In the two successful cases it was the other party who had taken on the hard work and the responsibility of making decisions The opinion that a little business is easy to run if the initial financial outlay can be covered is certainly mistaken; to make a small business pay demands hard work, sacrifice and long hours of personal attention

Forty (80 per cent.) of the patients put their newly acquired wealth into safe investments. Medically, this is probably the least useful of all the ways of using the money, for the interest, in most cases, derived from the investment is so small as to make little difference to one's standard of living.

My advice would be that patients should spend their money freely in seeking good health, in holidays and in improvements in their houses and in other amenities of living.

True intellectual impairments were exceedingly rare Patients who stated they were unable to work could play billiards and snooker and could add the complicated scores of these games with ease; they could also follow football results and were good judges of form. Few relatives were of the opinion that the injured had lost their wits When approached regarding their financial affairs the injured were very quick to appreciate any decision that affected their interests, either advantageously or otherwise. They are well aware that someone else is responsible for their disability On the other hand, if personality is an expression of intelligence, then intellectual deterioration of some form is a common sequel of a closed injury of the head.

Having equipped oneself with clinical information derived from the study of a case from every possible angle, an attempt has to be made to diagnose the pathological nature of the illness. This is a very difficult problem and one that in the past has been far too often neglected. Faults in diagnosis lead to unnecessarily prolonged morbidity and to waste of man-power My own suggestion regarding diagnosis is that an effort should be made to fit a patient suffering from residual post-concussional symptoms into one of the following groups:—

1 **Organic Dementia.**—In this type of illness the brain has been damaged in a physical sense The patient's disabilities are based on neuronal paralysis and are therefore inevitable and permanent *In such cases a patient must, at the time of accident have received* a severe physical injury to the brain which rendered him deeply unconscious for at least twelve hours The fewer a man's natural endowments the more easily is he incapacitated for competitive life and thus the pre-accident mentality of a man must be known as well as the severity of his brain injury before the diagnosis of organic dementia is made Only those patients who show frank intellectual as well as emotional deterioration should be placed in this group This illness leads to very tragic results and merits compassionate treatment and generous financial adjustments

2 **Psychoneurosis.**—Occasionally a state of true psychoneurosis is *precipitated by the injury By psychoneurosis is meant that* type of mental illness the underlying pathology of which is quite unknown, and which seems to come from within the very being of the patient to make him profoundly and persistently unhappy the whole time he is awake It is not an illness that has been precipitated by dissatisfaction or anxiety and is not cured by changes of environment or by adjustment of employment or of personal relationships The illness appears to be of a biological nature and is a psychiatric problem

3 **Substitution**—In this group the [illegible] illness is that of escape or of emotional [illegible] illness *to escape from uncongenial* [illegible] that he has always shouldered [illegible] sequences of misdoing He may [illegible] he is fond of and who he thinks are neglecting him This is the type of illness that is well within the capacity of any doctor to diagnose and to treat Indeed it is the duty of any doctor whatever illness he is treating to be aware of the type of reaction and to be willing to go to the trouble of guiding and helping the patient back to good health

4 **Psychological Illness**—To every physical illness there is a mental reaction of some kind In many cases the reaction is reasonable, *how far it is reasonable can only be determined by* one's own reaction to illness. In every case a decision has to be made whether they are a true reflection of his inner feelings The only cure in such cases is to remove the underlying illness if this is possible, for example, a persistent or oft repeated headache due to physical causes such as a chronic subdural hæmatoma or subdural adhesions can lead to depression lack of interest and loss of confidence Very great care must be taken before a patient, following a head injury is diagnosed as being merely neurotic

5. **Malingering.**—There are only two ways in which malingering can be diagnosed. One is by confession of a patient in complete possession of his wits and the other through espionage by a skilled and disinterested observer.

If the sequels of head injuries are to be minimised, patients must be adequately treated in the acute stages of their illness. For this, a sufficient number of surgeons, not necessarily neurological surgeons, will have to be specially trained in the subject of trauma of the head. One of the weaknesses in the past has been that far too many patients have been under the care of surgeons who were not interested in the problem.

Medical rehabilitation consists in a system of graduated mental and physical exercises carried out under favourable conditions and under the guidance of a doctor who fully understands the underlying problem (Fig. 259).

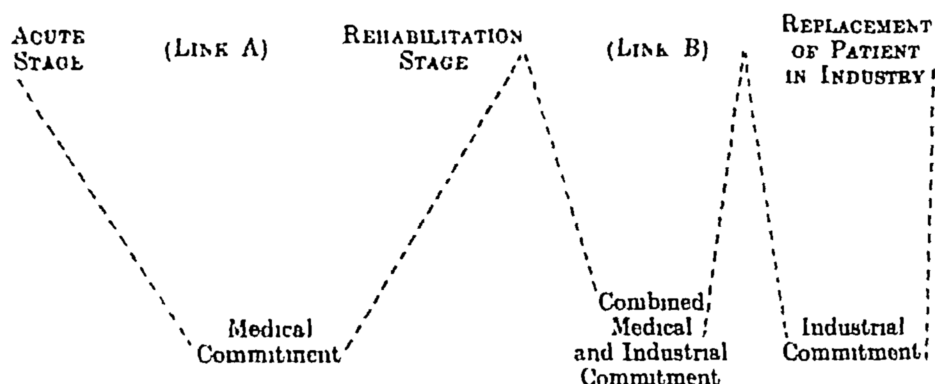


Fig 259

The link between the acute stage and the rehabilitation stage is an important one and there must be the closest co-operation between these two phases of treatment. Indeed, the doctor who is responsible for treatment in the acute stage must see his head-injured patient right through the medical rehabilitation stage until his patient returns to work. Rehabilitation and replacement in industry is being planned but, of course, its success to a large extent is governed by economic conditions. It is important that medical personnel in charge of the rehabilitation stage should go out into industry and make a study of the nature of the jobs that the men they have rehabilitated are supposed to go back to. Ample facilities are given in industry for this kind of observation and I myself have found it most interesting to visit the heavy industries of mining and of shipbuilding.

What I wish to speak about in particular is link B, the link between the medical phase of treatment and industrial replacement (Fig. 259). Efforts are being made by the government,

through the Ministry of Labour Offices to let medical practitioners know what kind of work is available for the injured It is true that the medical profession is being asked to fill in Ministry of Labour forms which will give detailed medical knowledge about patients who have been injured and who are seeking work Medical and industrial liaison is still, in many cases unsatisfactory and thus is largely a medical fault How can this liaison be improved? Let us assume that a group of patients has been adequately treated both in the acute and in the medical rehabilitation stages and that link A is a satisfactory one in other words let us suppose that there have been no medical faults up to the end of medical rehabilitation The patients that finally issue from the rehabilitation centre to be received in industry will be of different types and different grades of physical fitness and of mental intelligence To understand this final complex it is necessary to review the factors which act and interact to govern prognosis These factors are three in number and are as follows —

1 The nature and severity of the injury
2 The type of man who is injured
3 The nature of the job to which the man must return after convalescence

The tables on pages 466 and 467 express in diagrammatic form what I believe to be the influence each factor has on the final result of treatment

Patients in Table I will adequately take care of their own interests as soon as they are discharged from medical care How well patients in Table II fare in employment depends largely on prevailing economic conditions When jobs are plentiful men in this group on the whole return to work early and work satisfactorily It is Table III that remains a lingering medical commitment What can we do to curtail morbidity and to minimise waste of man power? To attain these objects I would like to suggest that within three months of an injury to a head a precise statement of the following details be laid before a small medical tribunal —

1 An assessment of the man s pre-accident endowments and character also an account of his working record

2 The nature and severity of the injury and in particular how deeply and for how long a man was rendered unconscious Details of any specific and irreversible damage such as the loss of an eye

3 The nature of a man s convalescence whether it was smooth or otherwise

TABLE I

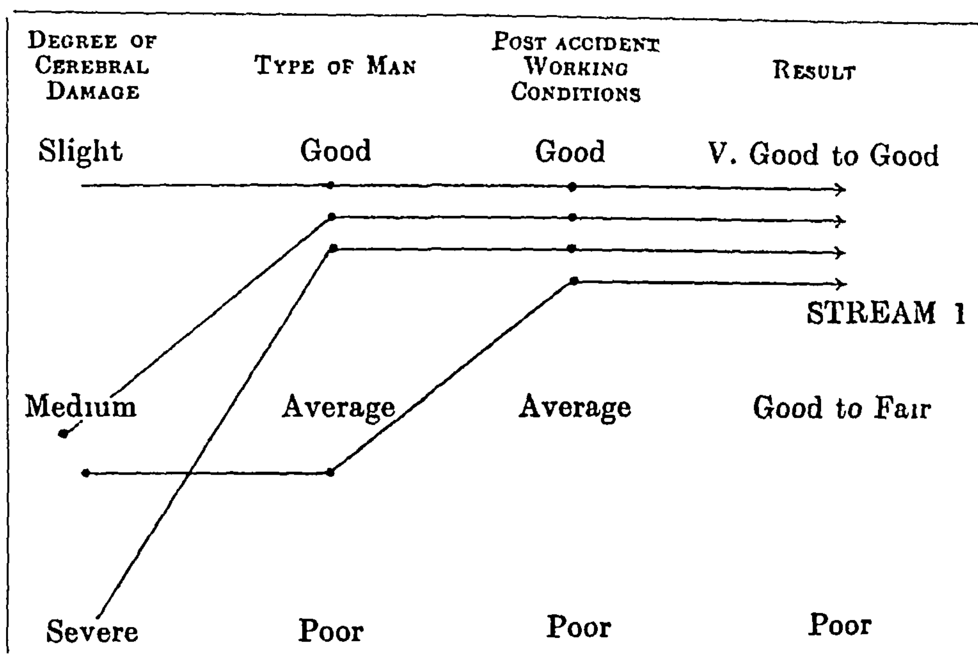


TABLE II

Degree of Cerebral Damage
Type of Man
Post accident Working Conditions
Result

Slight
Good
Good
V. Good to Good

Medium
Average
Average
Good to Fair

STREAM 2

Severe
Poor
Poor
Poor

TABLE III

Degree of Cerebral Damage	Type of Man	Post-accident Working Conditions	Result
Slight	Good	Good	Good to Good
Medium	Average	Average	Good to Fair
Severe	Poor	Poor	Poor

STREAM 3

Poor post-accident working conditions nearly always lead to a poor post-accident readjustment. In other words incentive has a dominant influence on prognosis. A sufficiently severe injury can permanently incapacitate the best of men, however favourable may be their post-accident conditions of working.

4 Whether a man needs further medical treatment and if so what kind and for how long.

5 Is the man fit for work? If not, why not?

6 If a man is not fit for his own work, is he fit for part of it? If he is not fit for part of his own work, is he fit for any other kind of work?

7 Few men are rendered incapable of doing any kind of work, but on those occasions when it is thought a man has been rendered permanently and completely incapacitated a firm statement should be made on this point. Until a final decision has been made regarding a man's ability to work and what kind of work he ought to do his case should be reviewed every two months.

There will always remain a residue of patients who refuse to work or who if compelled to work will work so badly that they are very soon discharged. For this residue medicine has no remedy, as it is impossible by medical means to compel the lazy to work; the attempt to make them work is not a medical commitment.

Whatever positive contributions my survey[1] of the residual illness has made to the post-concussional syndrome, one of its main results has been to emphasise the gaps in our knowledge. Much is known of the physics of injury and of the pathology and physics of recovery, on the other hand far too little is known of the machine that receives the damage. What is character and what is personality? How much is a man responsible for his behaviour? What credit is due to a man for his high endowments? Can an injury in fact damage a personality or can it merely remove an inhibition and allow an emergence of a basic character? Little indeed is known about these matters: probably the problem will never be solved by physics and pathology alone because some of the basic factors are of a biological or philosophical nature. The subject still remains to be studied from every angle.

[1] "The Long Term Results of Injuries of the Head" A medical, economical and sociological survey *Jour Ment Science*, 1949

INDEX OF AUTHORS
AND
GENERAL INDEX

INDEX OF AUTHORS

GENERAL INDEX

A

B

C

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